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ALCOHOL AS A FACTOR IN VICTORIAN ROAD COLLISIONS.

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Into every driver's mind as he hurtles towards an oncoming car at a comfortable seventy miles an hour there must at some time have flashed the thought: "Suppose this guy coming at me is drunk!" It's a playful little homemade thriller, a gruesome whimsy to relieve the moment's boredom, but the real joke, the hidden topper, is the considerable statistical chance that he is.

—With apologies to Bergen Evans.
The Spoor of Spooks.

THERE are a number of distressing features about the road collision problem in Victoria. First, the economic and human loss for a population of two and three-quarter million people using 760,000 motor vehicles is a serious one. Thus, in 1959, in Victoria, 666 persons were killed and 15,571 persons injured in the 27,697 separate motor-vehicle collisions reported to police (Figures I and II). For the year 1957 the cost in insurance claims alone was £12,100,000. Indeed, the whole Australian record of traffic collisions is an unenviable one, since it is the worst for the heavily motorized nations, and the worst in the world when mortality rates are considered on a population basis.

Secondly, there is a lack of data of all kinds in relation to motor-vehicle collisions in Victoria. Concomitantly, and possibly one of the main causes of this lack of data, there is the absence of both scientists and scientific method in the investigation of traffic collisions. This is in marked contrast to our methods of investigating aircraft accidents; our aircraft safety record is one of the best in the world. We lack in Victoria even the elementary data, such as how people die in collisions. We do not know how common is ejection from a crashed vehicle. At the moment we do not even know the makes of vehicles being involved. We cannot rely completely on American work, since many vehicles here are not present on American roads, the Holden car being a prime example. The resultant of this lack of scientific method is that road collisions are regarded as the province of police and lawyers, who, as it were, fight over the bodies, with the insurance companies barracking on the side. It should be noted that policemen are neither trained scientists nor statisticians, and that they investigate road collisions from the point of view of law-breaking. The insurance assessor investigates to determine negligence. Records produced by such investigation are of little help in the systematic determination of how the accident occurred, and of no help whatsoever in producing the reason why. Thirdly, we of the medical profession have been so occupied in treating the dying and the injured that, with isolated exceptions such as Dr. Ric Bouvier of Morwell Safety Town, we simply have not realized that the principles of prevention of occurrence and of progression which most of us practise every day in preventive medicine also apply to road collisions.

It matters little how people are being killed and maimed, whether by infectious disease or so-called accident, since it is the aim of preventive medicine, that science, in which "medicine joins hands with common sense", to prevent that killing and maiming. It is depressing to hear some practitioners adopt a defeatist attitude to road collisions—"they're inevitable, you'll never stop them"—when the principles already being applied effectively to industrial accidents can be applied to traffic collisions. After all, it is the human element which is predominantly at fault, both in industry and on the roads. The attitude of some members of the profession is even more difficult to understand when one considers the number of our colleagues already killed and injured on the roads.

Other features are the predominance of youth in our road casualties (55% of casualties are aged under 40 years). This leads to an incredible loss of working years for the community at a time when the nation is trying to build up its population.

The economic loss from road collisions in America has been estimated at 2% of the national income (McFarland, 1957). The Australian figure would be in proportion.

Lastly, despite our many amateur road-toll experts' views on bad roads, fast cars and poor lighting as being the main causes of road collisions, it is still human error that is responsible for 95% of the carnage.

The Value of Present and Projected Victoria Road Collision Statistics.

Similar statistics to those kept in Victoria have been designated in England as misleading and "rubbish" (Spriggs, 1958). Causation statistics are based on the opinion of the policeman on the spot, the majority of causes given being the immediate driving fault which precipitated the collision. Thus, as the statistician himself says, important antecedent causes such as fatigue, intoxicating liquor and inexperience receive little or no attention.

For example, there are many reasons why alcohol is overlooked both as a prime and as an antecedent cause. Death or injury in an accident usually precludes any mention of alcohol as a factor, because a corpse or hospital patient cannot be asked to do the peculiar manœuvres known by convention as a sobriety test. Almost invariably there is a time lag (or sobering-up period) between the occurrence of the collision and the start of police investigation. Such investigation only too frequently is a very cursory one. Post-mortem examinations are infrequently performed on collision victims, particularly in the country, while, if they are performed, blood alcohol levels are not estimated. Frequently one finds at the scene of collisions drivers who have taken more than enough alcohol to depreciate their driving efficiency considerably and obviously, yet they are not at the staggering, slurred speech stage.

The difficulties of the statistician may be shown by three examples:

1. A motor vehicle skidded from the middle of the road into a post, the driver's wife being killed. An "unofficial" blood alcohol estimation showed a level of 0·278%. The statistical cause was: "Car skidded on tram lines and driver lost control."

2. A pedestrian with a post-mortem blood alcohol level of 0·18% was hit and killed by a driver with a blood alcohol level of 0·15%. The statistical cause was "Careless walking and careless driving."

3. A driver was gaoled for six months for drunken driving; his blood alcohol level was 0·214%, and as a result another motorist was killed. The statistical cause was: "Other causes."

This, then, is the state of affairs which led Piret (1952) to state that there appears to be a conspiracy of silence between the authorities, the lawyers and the witnesses to wrap the effects (of alcohol on the roads) in a cloak of innocence. The statistics of causes at present and—since they are still to be collected by the policeman on the spot—in the future are for practical purposes useless. The only difference in the future collection of data is that the unfortunate policeman on the spot has now to fill in the answers to 250 questions. This will in a large number of cases be badly done or not done at all because of a severe shortage of men and time.

A Method of Obtaining Reliable Statistics.

McFarland (1957) has pointed out that the epidemiological approach developed for the study and control of infectious disease is especially applicable to road collisions. This epidemiological approach can fulfil the need for "a new and comprehensive approach to the control of collisions in which the relationships between the multiple factors in collision causation can be studied and classified". This approach must involve an interdisciplinary case study—that is, an intensive study of a few correctly sampled collisions by teamwork using the methods and techniques of as many disciplines as are required, statistics, engineering (both human and technical), physiology and psychology amongst others.

This approach unfortunately is not used and, indeed, has not even been officially suggested in Victoria; until it is used all remedial measures are simply stabs in the dark. One wonders why this is so. This type of approach could be got under way very quickly and cheaply, since it requires no big buildings or laboratories, while men with a knowledge of aircraft safety and engineering could be seconded by the Commonwealth Government to work on road problems. Incidentally, the Commonwealth has quite a defence problem on the roads, since many servicemen are being killed and injured in road collisions.

Lack of finance, therefore, is no argument against this approach.

Simple Epidemiology of Victorian Road Collisions.

One can obtain with a little effort figures of some value concerning road collisions distributions in Victoria. These immediately point to the unreliability of the official statistics indicting alcohol in about 2% of collisions and in 3·5% of fatal collisions.

First, the peak accident day of the week is Saturday. The traffic density on Saturday is generally below that on all other days in the week. Figure III shows that Friday is the next most "collision-prone" day. So-called Sunday drivers do not apparently cause the collision rate to rise on Sunday.

Secondly, Saturday is the worst day in the week for road fatalities (Figure IV). For interest, Figure V shows that Saturday is the day of the week on which police make most of their arrests for drunken driving.

Thirdly, when Victoria's road collisions are plotted on an hourly basis (Figure VI), two peaks are found, one major one between 6 and 8 p.m., and a second minor one between 11 p.m. and 1 a.m. The most obvious peaks are demonstrable on Saturday, but all days of the week show a similar pattern.

Fourthly, road fatalities plotted on an hourly basis show a similar pattern (Figure VII), while the times for arrest of drunken drivers by police show the same two peaks (Figure VIII). It should be noted that 70% of Victoria's drunken drivers (1500 per year) are found by police only after being involved in a collision.

These distributions at first are somewhat difficult to comprehend, since peak traffic on most roads is from 4.30 to 6 p.m. on week-days. However, when one realizes that Victoria's hotel bars are open only from 9 a.m. to 6 p.m. and are closed on Sunday, these graphs become explicable. A fifteen-minute period of grace is allowed from 6 to 6.15 p.m. to allow clearing of the bars. Most bars put on an admirable counter lunch—I have seldom seen a drunken driver within four hours of a meal. Licensed clubs may serve liquor till a late hour.

For comparison, two charts of English road collisions are shown. Hotels in England close at 10 p.m. Comment

is unnecessary (Figures X and XI). Seasonal distribution in Victoria shows a peak in early summer.

There is a very obvious geographical distribution of serious accidents in Victoria. In 1959, 426 persons were killed in country accidents, compared with 240 in Melbourne and its suburbs. It is a sad commentary that we know next to nothing about country accidents, except that

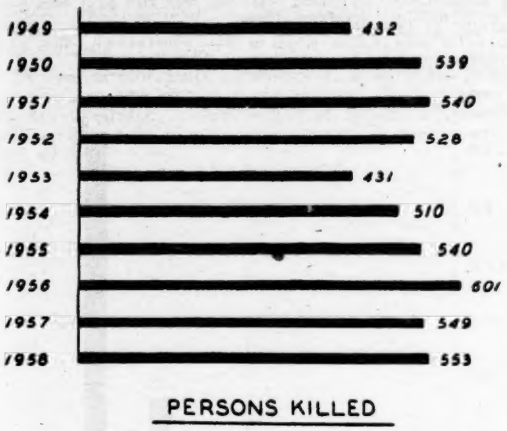

FIGURE 1.
Graph showing number of persons killed yearly on Victoria's roads; 666 persons were killed in 1959.

they occur at speed and that this speed does not kill many people between 6 a.m. and 4 p.m. Unofficial police and medical information from the country indicates that alcohol is responsible for many road deaths and injuries in the country.

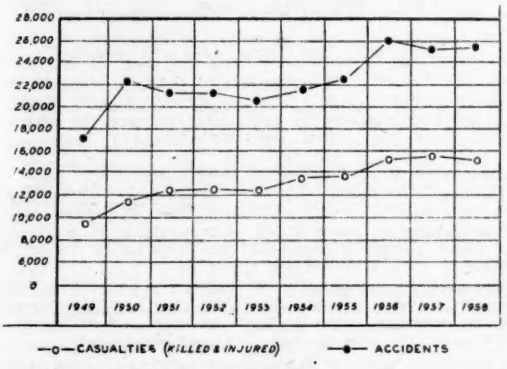

FIGURE II.
Total casualties and total vehicular collisions reported to police in Victoria since 1949.

It is frequently stated that the driver is not responsible for all the carnage on Victoria's roads, and this is so. However, in the year ended June, 1958, of the total Victorian casualty list, motor vehicle drivers were responsible for killing 378 persons and injuring another 9799 people. These figures represent 68% of the total casualties for the year.

THE INCIDENCE OF ALCOHOL IN VICTORIAN ROAD COLLISIONS.

The Literature.

In the last decade many papers have appeared in the literature, all indicating that, far from being a minor contributory cause, alcohol is a common and major contributory factor in road collisions. Only two papers have appeared on the subject in Australia — an odd fact for such a heavy drink-driving country.

Hindmarsh and Linde (1934) in Sweden found that 44% of injured drivers brought to hospital had been drinking: 29% of their injured drivers had blood alcohol levels greater than 0·1%. Gerber, of Ohio, quoted by

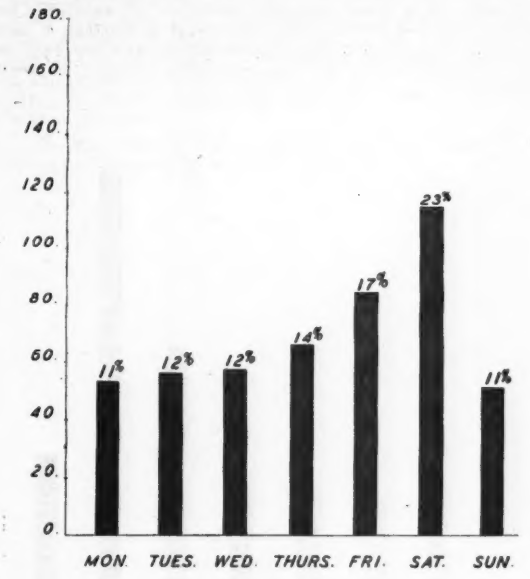

FIGURE III.
Victorian road collisions recorded graphically according to the days of the week on which they occur.

Plymat (1955), found that in 51% of his fatal road collision victim autopsies from 1941 to 1944, alcohol was found in the blood *post mortem*. Spain, Bradess and Eggston (1951) stated that 46% of all those killed in traffic collisions had a blood alcohol level over 0·08%. "Accident Facts" (1956) quotes a Delaware study over the three previous years, showing that 40·6% of all drivers involved in fatal collisions had been drinking.

Freimuth *et alii* (1958), in Baltimore, thoroughly examined 500 consecutive highway fatalities, death having occurred in each case within 12 hours of injury. In 241 deaths, i.e. 48·2%, alcohol was deemed a causative factor. Similar papers can be found emanating from France, Belgium, Britain, Germany, Canada and a number of other countries.

In Australia, Pearson (1957) in Perth, a city with a population of 396,000 people, estimated blood alcohol levels as a routine in his traffic collision autopsy victims from 1950. He did not take samples when survival was greater than 12 hours. Of the total 218 tests performed, 42·2% showed alcohol in the blood-stream, 39·4% of the 218 showing levels greater than 0·1%. Surviving drivers were not, of course, included.

Bowden *et alii* (1958), from Melbourne, estimate that at least 25% of all vehicle drivers killed in Melbourne have blood alcohol levels of 0·1% or more.

Victorian Hospital Experience.

It is difficult in busy casualty wards of any general hospital to get an accurate estimation of the number of road collision victims who have been drinking. However, checks have been made in the casualty wards of several hospitals. In one, 70% of all traffic victims were shown to have been drinking, including 56% of motor-car drivers. This check was made at about Christmas, 1957. Another check in another hospital in 1959, at a time of heavy

newspaper publicity against drink-driving, showed that 33% of traffic victims admitted that they had been drinking.

Casualty staffs at all Victorian major hospitals leave no doubt in one's mind of the importance of alcohol on the roads.

Mortuary Experience.

Whilst working at the Melbourne City Mortuary in 1956-1957, in a small personal series of 50 autopsies on traffic victims, I found alcohol present *post mortem* in half the subjects. At the inquest hearing in another 14% of cases it was found that the surviving driver had been drinking.

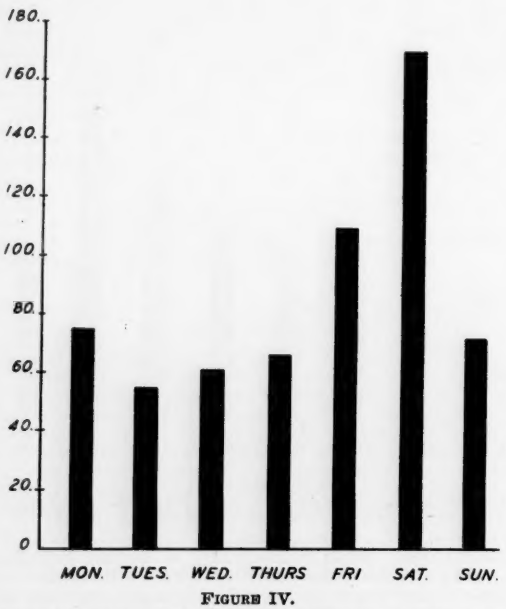

FIGURE IV.

Victorian road deaths recorded graphically according to the days of the week on which they occur.

This figure for the presence of alcohol in 64% of fatal collisions in either the offender or the victim (sometimes both) was cross-checked by examining a further 200 inquest files on traffic victims. Blood alcohol levels, when available, were in the main above 0·1%

The Experience of More Adequate Investigation.

The Victorian police several years ago established a six-man Accident Appreciation Squad as a pilot unit to assist in the investigation of fatal road collisions in and around Melbourne. In the first year of the squad's operations, 173 serious collisions were investigated, involving 125 deaths. (This represents 66% of the deaths which occurred in the city and suburbs for that year.) In 65 of the deaths alcohol was regarded by the squad as a main contributory factor, whilst alcohol was present in another five fatal accidents but was not thought to be contributory.

Subsequent experience of the squad has served only to confirm these figures.

Frequently, while driving from one collision to the next, I have found that it is difficult, particularly on Saturday nights, to find a collision in which alcohol is completely absent. Often, if the driver is sober, the pedestrian is drunk.

"Lost Week-Ends" in Victoria.

In an attempt to convey further the importance of alcohol in Victoria's fatal road collisions, the deaths for two ordinary week-ends follow.

On the first week-end four persons died, as follows:

A boy was killed on a bicycle when hit by a motor-car. The driver was arrested for drunken driving.

Two young people died when their car hit four trees. The driver's blood alcohol level *post mortem* was 0·164%.

A driver died when his car hit a tree. His blood alcohol content was 0·160%.

During the second week-end 10 people died, as follows:

A woman who had been drinking was run over and killed by a border-line drunken driver.

A child was killed when a car overturned. The blood alcohol content of the driver was 0·182%.

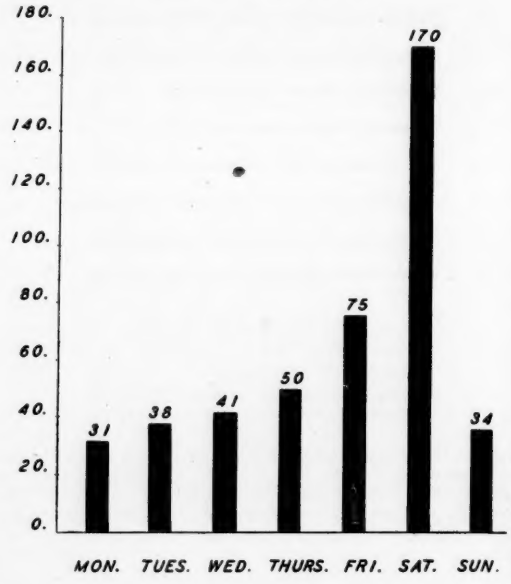

FIGURE V.

Number of Victorian drunken drivers recorded graphically according to the day of the week on which they are arrested.

Three died in a head-on collision, two others receiving fractures of the spine. The responsible driver *post mortem* was found to have a blood alcohol content of 0·278%.

Two pedestrians were killed while walking on a footpath. The blood alcohol content of the responsible driver was 0·15%.

Two people died when a car crashed into a truck. The responsible driver had been charged with dangerous driving and checked for his sobriety by police half an hour previously. He said he had only had six beers!

A boy was killed at 6.30 p.m. while walking along a country road. No details are available as to the sobriety of the driver.

The Serious Single-Vehicle Collision.

A recent important paper by Haddon and Bradess (1959) deals with alcohol in the single-vehicle fatal collision. Post-mortem blood alcohol levels were studied in a group of drivers whose fatal accident involved neither pedestrians nor other vehicles. Of the 83 subjects studied, 49% had blood alcohol levels above 0·15%, while in an additional 20% the blood alcohol level fell between 0·05% and 0·1%.

Over a period of several years I have collected a personal series of 54 serious single-vehicle collisions. Fifty-seven persons were killed in these collisions, 39 of those killed being passengers. In 17 cases the vehicle ran off the road and overturned, while in the other 37 cases the vehicle collided with stationary objects, such as trees, posts and correctly parked vehicles.

In nine collisions causing 14 deaths the driver had not been drinking. In the other 45 cases alcohol was present. In 40 cases the blood or breath alcohol content of the driver was over 0·1%. In four cases the driver's blood alcohol content was between 0·02% and 0·1%. In one case there was a history of a "pub-crawl".

The collisions generally occurred on good-quality straight roads or gentle curves. None occurred between 6 a.m. and 2 p.m., peak frequencies being after 7 p.m. and after 2 a.m. The maximum blood alcohol content in the series was 0·311%.

The series represents a number of catastrophes in which no one but the driver is to blame. It indicates that alcohol is present in over 80% of serious single-vehicle collisions and is a main contributory factor in at least 75% of cases.

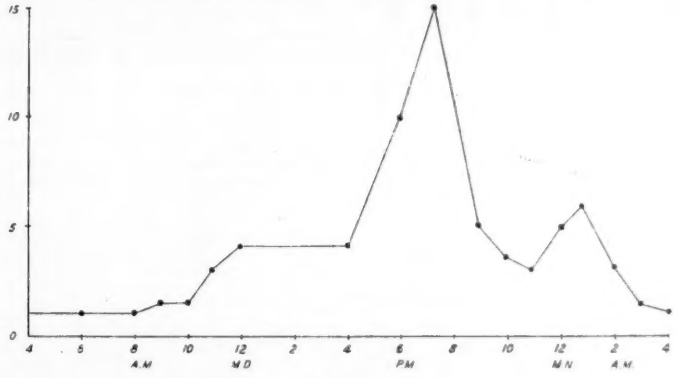

FIGURE VI.
Victorian road collisions on Saturday plotted on a time basis.

THE AMOUNT OF ALCOHOL IN TRAFFIC VICTIMS.

It is usually argued that drivers and accident victims admitted to hospital have only had a couple of beers; unfortunately, in my experience this is far from the truth. This is also in accordance with the experience of Spriggs (1958). One finds that the alcoholic driver (and his passenger) invariably underestimate his alcoholic consumption by a very considerable amount, the usual estimate being one-eighth to one-quarter of the minimum amount as indicated by chemical analysis. Not all are telling untruths, since in my personal drinking experience it is easy to lose count after only a few beers.

Table I shows the blood alcohol levels of a series of injured drivers going into hospital; none of these drivers was charged by police with the offence of drunken driving since they were injured. It can be readily seen that not one of this small series has had only a couple of drinks. Apart from the two drivers with a level of 0·06% some six or seven hours after the collision (in these cases the blood alcohol level at the time of the collision was obviously much higher), the minimum amount represented in this series is eight seven-ounce glasses of Victorian beer taken very rapidly. It is of some interest that several of these drivers have since sued in the civil courts as sober drivers.

THE TYPES OF ALCOHOLIC DRIVER.

The drivers under the influence of alcohol seen at collision scenes can be roughly graded into three groups (Figure IX). (i) The drivers in this group are frankly drunk, although, if they are injured sufficiently to require hospital treatment (even for a few stitches), a drunken driving charge may not be preferred by police because of the certainty of the legal defence of head injury (1508 charges were preferred in 1958). (ii) In the second group the drivers are border-line drunk, with some slurring of speech, poor balance and a bit of a stagger; usually, however, these drivers are experienced drinkers, and have the capacity to pull themselves together sufficiently to pass the rough sobriety test. Many cases of dangerous driving (2323 charges in 1958) and lesser traffic offences apply to this type of driver. (iii) In the third group the drivers have bloodshot eyes, a strong smell of liquor, and supreme confidence; they are often argumentative, but are completely unable to explain either the circumstances of the collision or their bad driving. This type of driver almost always has an obvious lateral nystagmus indicating a minimum blood alcohol level of 0·05% to 0·06%. This type of driver is far more common than the first two groups combined.

One other point is that the drivers in all three groups are experienced and practised drinkers.

From the foregoing it can be seen that alcohol is present in quantity in the blood-streams of many vehicle drivers involved in road collisions in Victoria.

THE SIGNIFICANCE OF ALCOHOL IN VEHICLE DRIVERS.

Two questions immediately arise in so far as the car driver is concerned: (*a*) Is the alcohol significant? (*b*) If it is significant, how much alcohol is required before road behaviour is influenced?

There can be no doubt that in the higher ranges of blood alcohol level (above 0·15%) the alcohol is significant even in the case of so-called "good drinkers". Above 0·2% practically everybody shows the symptoms and signs of acute alcoholic intoxication. For example, the average blood alcohol content of some 1000 drunken drivers seen by myself over two years is 0·230%. However, there is now considerable experimental and statistical evidence in the literature that small amounts of alcohol have a considerable (and measurable) detrimental effect on driving skill.

The Properties and Effects of Alcohol.

It is unfortunate that those properties which make alcohol an ideal material to facilitate social intercourse are just those properties which are inimical to the driving and complete control of a motor-car.

The physiological effects of alcohol which are of importance in car driving may be briefly summarized thus (Drill, 1958): (i) It slows reaction time. (ii) It depreciates vision, and, most important, it reduces tolerance to glare. (iii) It depreciates coordination. (iv) It removes inhibitions, thereby leading to increased risk-taking, the individual responding to impulses which are ordinarily repressed. (v) After very little alcohol the drinker sees the world and himself through rose-coloured spectacles—the clinical state called euphoria. As Schmiedeberg (1902) put it:

In the psychic sphere there is first lost the finer grades of attention, judgement, reflection and ability to understand. The soldier becomes more courageous since

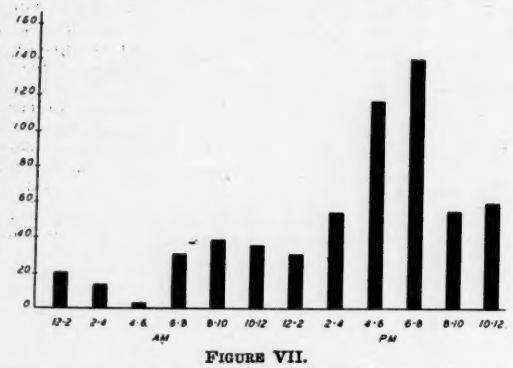

FIGURE VII.

Victorian road deaths recorded graphically according to the times at which they occur.

he observes the danger less, and reflects upon it less. The speaker is not tormented and influenced by the proximity of the public. He, therefore, speaks more freely and with more animation. One's self-appraisal rises greatly, often one is astounded at the ease with which one expresses one's thoughts and with the keenness of one's judgement on matters which are beyond one's mental sphere when sober and is later ashamed of this delusion.

The last effect is probably the most important, since it means that one is unable to realize, shortly after beginning drinking, that the other physiological actions of alcohol, such as slowed reaction time, loss of coordination, impaired vision and so on, are developing.

The Effect of Alcohol on Driving Ability.

As far as the demonstrable effect of alcohol on driving ability is concerned, several recent papers demand mention.

The first and definitive paper by Drew *et alii* (1958) aimed "to investigate the effect of small doses of alcohol on a complex skill resembling driving and to relate any changes found to the level of blood alcohol". These authors recognized that the method of measuring any change was of great importance, the subject under test frequently compensating for a short time for conditions producing an over-all reduction in his efficiency. For this reason, short single tests may fail altogether to detect any deterioration and may even show above normal efficiency. They suggested, therefore, reasonably long-lasting tests unless the deficiency in performance was a gross one. (It has been a common experience for me in testing the sobriety of obviously drunken motorists, to find that they can perform single tests perfectly.)

In using a dummy car, "the Miles motor driving trainer", for their experiments, they compromised between a complex continuous task of reasonably long duration, and one which did not become too boring. In the apparatus, a road scene is depicted on a screen, and the car appears to progress along the road and has to be steered by the driver. The test is devoid of danger and emergencies. Various amounts of alcohol were given to 40 volunteers of an average age of 31 years, the largest amount being three pints of average English beer or five fluid ounces of whisky per an eleven-stone man. They examined blood, urine and breath samples at various times after each dose. The experiment was controlled.

Road Behaviour under the Influence of Alcohol.

Drew *et alii* found the following effects from alcohol in the dosages mentioned: (i) The subject tended to veer from the correct side to the crown of the road. (ii) Other steering movements were affected. The subjects could not maintain a steady steering wheel; they wobbled and had collisions with the kerb. They had difficulty with corners, usually making too much movement of the wheel to negotiate the corner accurately. (These defects in driving skill can actually be observed on roads all over Victoria from 6.15 p.m. onwards every night, except Sunday. Many times I have observed cars continually wandering over the road, often on the wrong side of the centre line, suddenly veering back to the kerb on the correct side. When stopped by police the driver, is frequently not even "border-line drunk"; yet he is completely unaware that his driving has been anything but perfect. These defects obviously will account for a big proportion of road accidents, particularly when aggravated by any sort of emergency.) (iii) The accuracy of steering decreased progressively as the blood alcohol content increased, even with the small amount of alcohol used in the experiments.

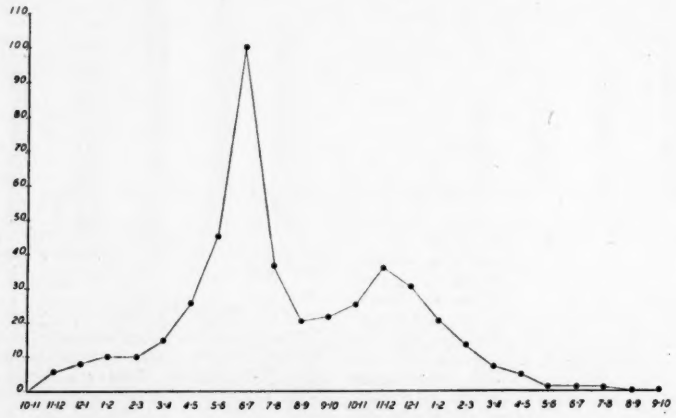

FIGURE VIII.

Graph showing the times of arrest of a series of drunken drivers.

Drew *et alii* concluded that impairment of driving performance paralleled the blood alcohol level. The maximum blood alcohol level reached was only 0·08% (80 mg. per 100 ml. of blood), and the average deterioration in driving performance was 16%.

This impairment in performance in practice means an increased likelihood of accident. After drinking, not only will drivers become involved in greater hazards, but also they will become less efficient in dealing with them.

Risk-Taking.

A second experiment, reported by Cohen, Dearnaley and Hansel of Manchester in 1958, aimed at assessing the effect of alcohol on the more complex psychological processes and performances involved in risks which drivers take, and in hazards which they incur. This was attempted because of conclusions, from various experiments involving reaction time, braking and steering skill, that because no changes were recorded after small quantities of alcohol, road safety was unaffected.

Cohen *et alii* make the point that it cannot be inferred that a driver is not a menace on the road because his reaction time appears normal or because a test of skill shows no impairment. The decisive feature is not the driver's skill in itself, but his skill in relation to what he believes he can do and what he will in fact undertake. The experiment consisted of giving experienced bus drivers whisky, and then obtaining the driver's estimate of his success at driving a bus through gaps of varying width. Having made his estimates, he then drove the bus through particular gaps.

Cohen *et alii* found that the trustworthiness of a man's judgement of his driving skill was impaired even after small quantities of alcohol, producing blood alcohol concentrations below 0·05% (50 mg. per 100 ml). The drivers who took alcohol became involved in greater hazards than alcohol-free drivers. The authors also found that the performance of the drivers, as well as their judgement, progressively deteriorated as they consumed more alcohol.

Because the deterioration of judgement and skill occurs at such low blood levels, Cohen *et alii* suggest that alcohol be forbidden for a specified period before driving. (It may be mentioned here that those safety-conscious people the airlines lay down in regulations an alcohol-free period before their pilots fly—a measure which meets with both public's and pilots' approval). The authors do not consider the interval 0% to 0·05% as safe. Their last conclusion is an important one, and needs no comment: "Alcohol intensified any driver's tendency to overrate his ability in relation to his performance."

Neither Drew nor Cohen found a threshold effect, both finding that performance started to deteriorate at very low blood levels.

Loomis and West (1958) also used a simulated car-driving apparatus to evaluate the effect of alcohol as it pertains to several of the functions involved in driving and controlling a car. They demonstrated a direct relationship between the blood alcohol level and the extent of impairment of function. All subjects showed impairment of performance at 0·05%, while in the presence of a blood alcohol concentration of 0·15% performance had decreased to approximately two-thirds of the control value.

Coldwell *et alii* (1958) in essence repeated Bjerver and Goldberg's work of 1950 with almost identical results,

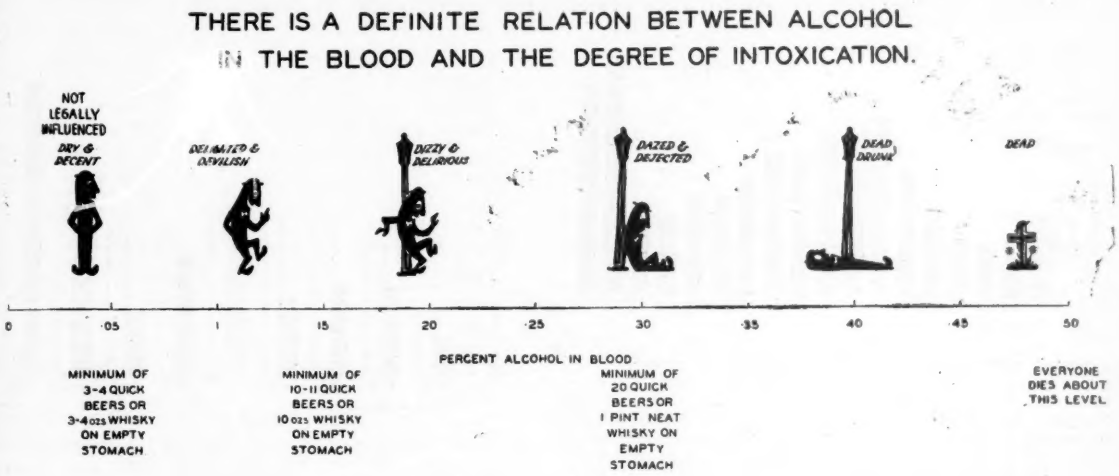

FIGURE IX.

Chart correlating clinical appearance with the blood alcohol level.

using actual driving tests. The beverage used was distilled spirits given to a group of 50 experienced car drivers. Under their experimental conditions, impairment of driving skill was obvious in one subject at a blood alcohol level of 0·036%. Signs of impairment were observed in five out of seven subjects with blood alcohol levels below 0·05%. Heavy drinkers showed less impairment than light drinkers at the same blood alcohol level, but eight out of 10 heavy drinkers exhibited signs of impaired driving skill at various blood alcohol levels varying from 0·05% to 0·12%. No driver retained his non-drinking driving skill at a blood alcohol level approaching 0·15%. Statistically, half of the 50 drivers tested exhibited significant impairment of driving skill at a blood alcohol level of 0·078%. Car-handling ability (as distinct from the ability to avoid hitting objects) deteriorated considerably in most drivers even at relatively low venous blood alcohol levels.

Deficiencies of Driving Tests.

However, Coldwell *et alii* note that the tests used (backing, parking and turning) were unlike practical driving in the following respects: (*a*) Speeds were very low. (*b*) There were no distractions from passengers. (*c*) No sudden emergencies arose requiring quick decisions. (*d*) Each driver through practice knew what to expect. (*e*) The attitude of the subjects was that of competitors

in a game, most making a definite effort and endeavouring to show that alcohol had no effect on their driving ability. These authors conclude that under actual driving conditions, particularly in urban areas, the effect of alcohol on driving performance would be more pronounced than that observed in their experiments.

The Hazard of Drinking, then Driving.

There are three other papers in the literature which, from actual observations on the roads, confirm the experimental work referred to above.

The first paper by Holcomb (1938) made a study of 207 drinking drivers involved in personal injury accidents, compared with 1,750 drivers in the general road population. He found that there was an equal percentage of drinking drivers in the accident and the general driver population group at a 0·05% blood alcohol level, indicating that at this level alcohol was not necessarily a significant cause of accidents. He also found that, as the blood alcohol

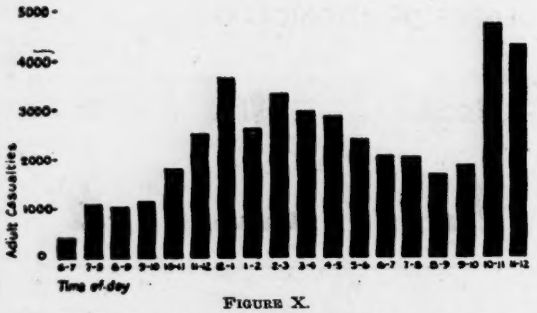

FIGURE X.
Graph showing times at which casualties occur on a Saturday in England.

content increased, the number of drinking drivers appearing in the personal injury accident group increased out of all proportion over the number of drinking drivers in the general driving group.

Holcomb discusses two other important points in relation to road accidents. The first is the question whether darkness is really the cause of accidents at night. If this was so, an accident curve related to the traffic density should be uniform. Holcomb found that this was not so. Secondly, he discusses fatigue as a further cause of accidents, but argues that it would be impossible for fatigue to account more than superficially for the increase in accidents associated with alcohol. (It may be added here that fatigue officially is responsible for less than 1% of Victoria's accidents; this is probably a considerable understatement.) Holcomb adds that the fatigued driver is more likely to compensate for his disability than is the drinking driver. He can recover much more readily from fatigue than from the effects of alcohol.

Lucas *et alii* (1953) compared the blood alcohol levels of 433 drivers involved in accidents with those of 2015 drivers passing those accidents. Their conclusions were as follows: (*a*) The hazard was significant when the blood alcohol level was above 0·1%. (*b*) In the range 0·1% to 0·15%, the hazard of accident was two-and-a-half times that when the concentration in the blood was less than 0·05%. (*c*) The hazard of accident with blood alcohol levels of 0·15% was approximately 10; that is, there were 10 times as many drivers with blood alcohol levels of 0·15% and above in the accident group as in the control group of uninvolved drivers.

Smith and Popham (1951) estimated the blood alcohol levels of 542 of 919 drivers involved in personal injury collisions. They compared the levels of those rated highly responsible for the accidents in which they were involved with the levels of those rated as either not responsible or only slightly so, and demonstrated an increasing statistical excess of collision-prone drivers which first became significant in the 0·03 to 0·05% range.

A Note on So-Called Tolerance to Alcohol.

It is a *sine qua non* of any discussion amongst lay people, and surprisingly many medical practitioners, that the good, experienced drinker does not get into trouble on the roads. This proposition, in my experience of over 1000 drunken drivers and some thousands of road collisions, is a most dangerous one. Most drivers in trouble for drinking and driving, whether arrested for drunken driving or seen at a collision, are good, experienced drinkers, and all seem to practise assiduously at drinking. Unfortunately some social prestige attaches to this.

Goldberg (1955) in Sweden, using controls, showed that chronic alcoholics and other alcohol abusers were heavily over-represented in a consecutive series of 2100 persons convicted of drunken driving. (In Sweden this refers to a driver with a blood alcohol level greater than 0·08%. In Victoria the average convicted drunken driver has a blood alcohol level of about 0·23%.) Further Bjerver

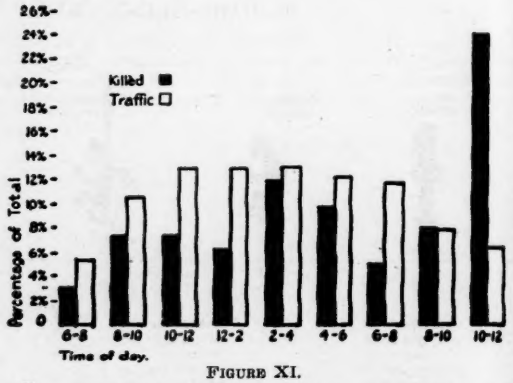

FIGURE XI.
Graph correlating drivers killed on a Saturday in England with the traffic flow.

et alii (1955) showed, in a study on 71 road collision victims in hospital, that although heavy drinkers were highly over-represented amongst those casualties who had been drinking they were not over-represented amongst those casualties who had not been drinking. This suggests immediately that the changed liability exhibited by the heavy drinker groups was closely associated with, if not directly caused by, the use of alcohol.

Loomis and West (1958) sum the position up well:

> For medico-legal purposes there are no objective data to indicate that the confirmed alcoholic is or is not capable of operating a motor vehicle in a more nearly normal fashion than does the occasional drinker when both have an equal blood alcohol concentration. Certainly from the point of view of society there is no reason for giving the frequent drinker legal permission to drive an automobile with alcohol in his blood simply because he drinks frequently . . . . Should legislation regarding alcohol and traffic safety permit the frequent consumer of alcohol or the confirmed inebriate to drive on the assumption that he is a better driver to start with?

CONCLUSIONS AND SUMMARY.

It is general experience among "motorized" nations, despite "official" statistics to the contrary that alcohol is commonly present in considerable amount in the bodies of road-collision victims. This fact is not obvious to the public by virtue of inadequate statistics and insistence on the further production of those statistics.

In Melbourne the percentage of road collision victims smelling of liquor when admitted to casualty wards in general hospitals varies from 30 to 70. No data are available from country districts; but individual medical and police opinion would indicate that the percentage is high in the country.

At the Melbourne City Mortuary some 64% of road collision deaths are associated with alcohol, pedestrians, drivers, passengers, cyclists and other road-users frequently being found to have high blood alcohol levels *post mortem*. Blood alcohol levels estimated on unselected drivers being admitted to hospitals in Melbourne indicate generally the consumption of large amounts of liquor.

The Victoria Police Accident Appreciation Squad, in a series of fatal road collisions involving 125 deaths, found alcohol present in 70 cases. In 65 cases the alcohol was regarded as contributory, no blood alcohol estimations being available for the other five subjects.

The simple epidemiology of road collisions in Victoria can be explained only on the basis that alcohol is playing a large part therein.

In a series of my own of 54 single-vehicle accidents resulting in 57 deaths, alcohol was present in 45 cases; in 40 cases the driver's blood alcohol level was greater than 0·1%.

TABLE I.

Showing Blood Alcohol Levels of a Series of Drivers Admitted to Hospital after Collisions.[1]

Cause of Accident.	Blood Alcohol Content.
Driver hit tram head on	0·274%
Driver hit taxi head on	0·142%
Driver hit safety zone	0·275%
Driver side-swiped tram (1 dead)	0·220%
Driver rolled car	0·150%
Motor cyclist hit car head on	0·174%
Taxi driver refused right of way	0·115%
Driver turned in front of oncoming traffic	0·250%
Driver hit car head on (1 dead)	0·115%
Driver hit parked truck at 80 m.p.h. (1 dead)	0·175%
Driver hit car head on	0·185%
Driver hit post on wrong side	0·120%
Driver hit car head on (2 dead) (blood taken six hours after accident)	0·06%
Driver skidded and hit post (1 dead)	0·278%
Driver hit concrete wall beside road	0·173%
Driver hit post, then another car (1 dead) (blood taken seven hours after accident)	0·064%
Driver hit telegraph pole (1 dead)	0·162%
Driver rolled car (1 dead) (blood taken four hours after accident)	0·125%
Driver hit parked truck (2 dead)	0·268%

[1] None of these was charged by police with the offence of driving under the influence of intoxicating liquor. All stated that they had "only had a couple of beers". (0·015%—a minimum of one seven-ounce glass of Victorian beer or one ounce of whisky.)

The types of alcoholic driver are discussed, the drinking and border-line drunken driver being responsible for many more deaths and injury than the true drunken driver.

The significance of alcohol is discussed. Most of the work has been done overseas; but as the Australian is still a member of the species *Homo sapiens*, the world-wide conclusion that alcohol is deleterious to driving applies to the Australian drinking driver with at least equal force.

Road behaviour on both clinical and experimental grounds begins to be influenced in the blood alcohol range 0·03% to 0·05%, and is markedly affected at 0·15%. Obviously, any attack on road collisions must be spearheaded by an attack on alcohol; but a problem made up broadly of 50% alcohol, 35% bad driving and 15% mechanical defects, natural disease and other miscellaneous causes, needs a broad-front approach, based on accurate case study of carefully sampled collisions.

In the words of a neurosurgeon, G. F. Rowbotham:

> How can we get at the truth of this knotty problem—probably by one method and one alone; by a forum of a cross-section of the community of honourable men who faithfully will discuss the problem in terms of themselves. If most men were to speak honestly they would vote in favour of the opinion that a man drives his best when he has had no (alcoholic) drinks.

ACKNOWLEDGEMENTS.

While the opinions expressed in this paper are my own, the paper itself would have been impossible save for active help from many people (and perhaps for the stimulus provided by some others who cannot or will not acknowledge that there is an alcoholic problem on our roads). I am grateful to many conscientious and enthusiastic members of the Victoria Police Force who, despite pressure of work and frequently under great difficulty, were always willing to help. I am particularly grateful to the Photographic Studio, the Drawing Office, the staff of D24 and the Transport Section, as well as to the members of the Accident Appreciation Squad. I am greatly indebted to Miss Norma Byron and her staff of willing typists for their capable and efficient help.

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ADDENDUM.

Since this paper was written, a British Medical Association Sub-Committee on "Alcohol and Road Accidents" has published (*Brit. med. J.*, January 23, 1960) a full report of its findings. I agree completely with the committee's conclusion that the number of road accidents caused by alcohol has been "very considerably under-estimated". The committee considers the level of alcohol in the tissues a most valuable guide, since it may be quite high and may result in abnormal driving while the driver shows few clinical signs. The committee regards a blood alcohol level of 0·05% as the highest compatible with road safety generally.

The findings of this English sub-committee are essentially similar to those of a Road Accident Sub-Committee of the

Victorian Branch of the British Medical Association some 18 months ago. Both committees considered that the introduction of a law making it illegal to be in charge of a car with more than a certain level of alcohol in the tissues would considerably reduce the road collision rate.

Finally, it is interesting, in view of the amount of alcohol in Victorian drivers, to read that the English committee "cannot conceive of any circumstances in which it could be considered safe for a person to drive a motor vehicle on the public roads with an amount of alcohol in the blood greater than 150 mg./100 ml".

CEREBRAL ABNORMALITY AND THE ELECTRO-ENCEPHALOGRAM IN RELATION TO PSYCHIATRY.

PART I: THE VALUE OF ELECTROENCEPHALOGRAPHY IN THE DIAGNOSIS OF CEREBRAL ABNORMALITY ASSOCIATED WITH PSYCHOLOGICAL MANIFESTATIONS.

By K. ANDERMANN,
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DURING a review of 1000 electroencephalographic tracings of psychiatric patients, it was thought that the traditional approach of their interpretation in exclusively neurological terms was unsatisfactory. Electroencephalographic tracings supply information more valuable in psychiatry than is generally realized. I gradually became convinced that it was of paramount importance to consider psychopathological information, as well as neuropathological electroencephalographic evidence, in order to be able to understand the whole psychiatric picture.

Electroencephalography is a method by means of which minute electrical impulses originating in the brain are picked up on the scalp, amplified and traced on a moving sheet of paper. Normal subjects emit several electric rhythms which are popularly called brain-waves.

Most cerebral pathological conditions grossly distort the normal brain-wave pattern, so that the electroencephalogram can be effectively employed in the diagnosis of abnormalities such as epilepsy and space-occupying lesions. That the electroencephalogram is also of diagnostic value in psychiatry is, however, not generally realized. During the last decade the concept of epilepsy was greatly enlarged so as to include clinical states beyond major seizures and *petit-mal* lapses. This enlarged concept of epilepsy embraces not only psychomotor seizures, but also various partial seizures and localized epileptogenic foci, the clinical manifestations of which can frequently be controlled by anticonvulsant drugs (Gastaut, 1954; Penfield and Jasper, 1954). Discoveries of the psychological function of various cerebral structures, particularly in the temporal lobes, were valuable contributions to psychiatry, as abnormality in those structures often produces deviatons of the normal electroencephalographic pattern.

Statistics published in various countries suggest that the incidence of cerebral abnormalities responsible for psychiatric disorders in the general population is by no means negligible.

With regard to epilepsy, I made a computation based on British (W.H.O., 1955), Dutch (Ledeboer, 1955) and American (Gibbs, 1952) statistics. They suggest that about one in 200 members of the general population is an epileptic suffering habitually from major seizures, about one in 700 suffers from major seizures and in addition from epileptic psychological disturbances, and about one in 1500 suffers from epileptic psychological manifestations as the sole expression of the illness. This last group can often be dagnosed by means of electroencephalography only. In the general child population, the incidence of epilepsy, which seems to be mainly of the non-convulsive type, is thought to be even higher, of the order of one in 17 children (Thom, 1942).

The present paper is divided into two parts. The first part describes psychological abnormalities produced directly by cerebral pathological conditions. The second part discusses how cerebral pathological factors and psychogenic emotional factors may affect each other.

Clinical Material.

An analysis of 1000 consecutive electroencephalographic recordings taken at Mont Park showed that 48% of the psychiatric patients had abnormal electroencephalographic patterns, 15% had borderline patterns and 37% had patterns judged as lying within normal limits.

Epilepsy Directly Responsible for Psychopathological Manifestations.

The manifestations of some epileptics are so obvious that the nature of the illness can be readily recognized on clinical grounds alone. In other subjects, however, it happens that one single symptom, such as furor, lack of attention or hallucinations, dominates the entire clinical picture without any additional clue to its epileptic nature.

TABLE I.
Psychological Epileptic Manifestations Without a History of Major Seizures Among 1000 Mentally Ill Subjects.

Epileptic Psychological Manifestations.	Positive Diagnosis: Number of Cases.	Probable Diagnosis: Number of Cases.
Psychomotor epilepsy	4	3
Epileptic hallucinations and illusions	6	4
Epileptic somatic pain and dizziness	3	2
Pyknolepsy	4	0
Total	17 (1·7%)	9 (0·9%)

The Classical Form of Psychomotor Epilepsy.—The epileptic psychomotor seizure is characterized by a combination of emotional outbursts, automatic movements and subsequent amnesia for the seizure. The emotional outbursts may consist of laughing, crying, fear and aggression. The automatic movements vary from simple movements like smacking of the lips, blinking of the eyes, or fiddling with buttons, to integrated behaviour like undressing, confused talk, breaking furniture, attempting to run away and assaulting other persons (Gibbs, 1952; Hill and Parr, 1950; Schwab, 1951). Electroencephalographic tracings taken during a psychomotor seizure show that the normal brain-waves are replaced by synchronous bursts of theta waves in all cortical regions. In between the clinical seizures, the electroencephalographic recordings from about 88% of the psychomotor epileptics show spikes, or bursts of slow waves, in or around the anterior temporal regions.

CASE I.—This case was one of epileptic psychomotor "black-outs" without any other epileptic manifestations. A single, slightly dull woman, aged 42 years, suffered from episodes of "black-outs" during which she was not conscious over a period of 12 months. During one of these "black-outs" she took an article from a department store in such a clumsy way that she was easily apprehended by a floor-walker. The magistrate before whom she was brought sent her to a psychiatrist for his report on the "black-outs". The psychiatrist had the impression that she was suffering from simple schizophrenia. There was no history of major seizures. The electroencephalographic tracings showed high-voltage spikes and bursts of theta waves in the right temporal region. The bursts of theta waves had a tendency to spread synchronously to all other regions. No "black-outs" occurred after treatment with anti-convulsive drugs was started. (See Figure II.)

Epileptic Hallucinations and Illusions.—Psychological phenomena, such as hallucinations and a sense of unreality, may occur in auras preceding major seizures. Occasionally, there are subjects who experience psychological episodes of the nature of epileptic aura though they may have never

suffered from a major seizure. Williams (1956) observed, *in the majority of his patients with epileptogenic foci* situated in the anterior temporal lobe manifestations of abnormal sensations of fear, and he and Weil (1955) describe depressive states associated with epileptogenic foci in the temporal lobes. Gibbs and Gibbs (1952) found that epileptic seizures starting in the thalamic or hypothalamic structures could produce auras of pain, rage, hallucinations and disorders of sleep.

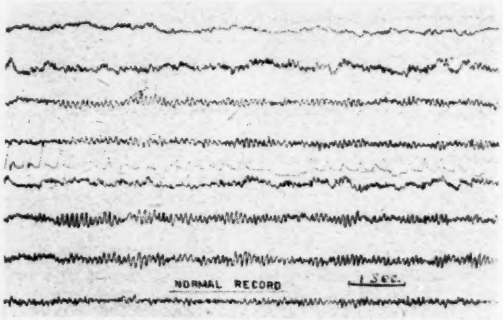

FIGURE I.
Normal electroencephalographic record.

CASE II was a case of epileptic olfactory hallucinations without any other epileptic manifestations. An intelligent professional man, aged 39 years, had for the past eight years suffered from brief disturbing episodes during which he "smelled something burning", lost temporary control of his movements and slipped to the left side. There was no history of major seizures. The resting electroencephalographic tracings were normal. However, after activation with 12 ml. of "Megimide" given intravenously combined with photic stimulation, his arms and head jerked and he was "smelling crackers". At the exact time when the jerk

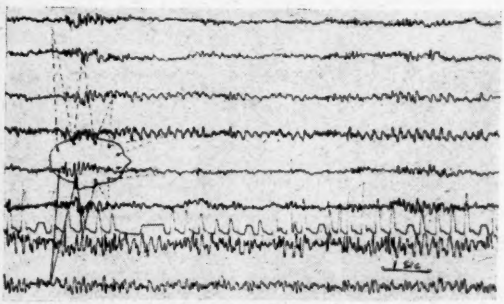

FIGURE II.
Spikes and bursts of theta waves in right temporal region.

and olfactory hallucination occurred, the electroencephalographic tracings registered synchronous spike discharges in both frontal-parietal regions. The olfactory hallucinations and the myoclonic jerks were controlled with "Dilantin". (See Figure III.)

Penfield and Jasper (1954) and Gastaut (1954) observed olfactory hallucinations in subjects with epileptogenic foci in the temporal lobes.

CASE III.—This case was one of epileptic acoustic hallucinations with behaviour disturbances, but without any other epileptic manifestations. A woman patient, aged 26 years, was suffering from episodes of hysteroid furors which, she said, were preceded by whistling and buzzing sounds. These turns occurred about once a year. The electroencephalographic tracings showed the presence of sharp waves and spikes, followed by a slow wave, in both posterior temporal and left parietal regions. (See Figure IV.)

Acoustic hallucinations, as a result of temporal epileptogenic foci, have been observed by Penfield and Jasper (1954) and Gastaut (1954).

CASE IV.—This was a case of visual illusions in a boy, aged 14 years, with a withdrawn and inhibited personality, who occasionally had the illusion that objects looked smaller than they really were. The general clinical condition resembled a chronic schizophrenic reaction or gross neurotic inhibition. There was a history of head injury at the age of two and a half years followed by occasional major seizures

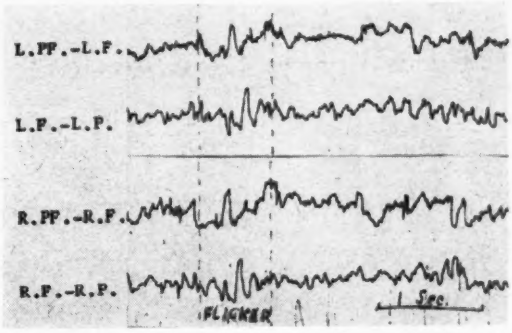

FIGURE III.
L.PF.: left prefrontal; L.F.: left frontal; R.PF. right prefrontal; R.F.: right frontal; L.P.: left parietal; R.P.: right parietal; R.A.T.: right anterior temporal; L.L.F.: left lateral frontal; L.M.F.: left midfrontal; R.M.F.: right midfrontal; R.L.F.: right lateral frontal.

for the subsequent 12 months. There was no history of major seizures after the age of three and a half years. The electroencephalographic tracings showed high-amplitude spikes, some of them followed by a slow wave, at the junction of the right temporal, parietal and occipital lobes. There were also poly-spike-wave seizure patterns. The electroencephalographic tracings were highly suggestive

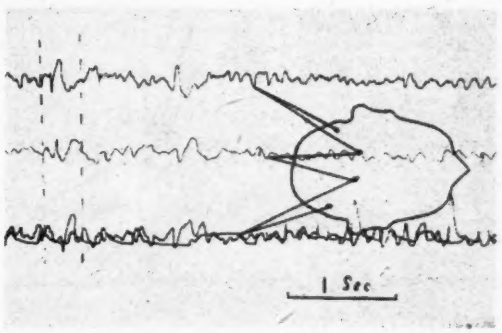

FIGURE IV.
Parieto-temporal epileptogenic focus.

of an epileptogenic focus at the junction of the right temporal, parietal and occipital cortex (Gastaut, 1954). Anticonvulsive drugs, to which small amounts of amphetamine were added, improved the patient's clinical condition dramatically. (See Figure V.)

Epileptic Somatic Pain and Dizziness.—**Many forms of epileptic seizures associated with primitive sensations or functions have been described by various workers (Penfield and Jasper, 1954; Gibbs, 1952).**

CASE V.—This was a case of epileptic gastric pain. A boy, aged 13 years, had been suffering for one year from gastric attacks. For the same period he also had presented a behaviour problem. The attacks consisted of gastric pain and nausea, though vomiting did not occur. Later, in school, he noticed that after such a "gastric attack" his right leg jerked for about one minute. A few days after that incident

he had a minor seizure on the playground. Hyperventilation produced theta-wave seizure patterns, and spikes in the left frontal region. (See Figure VI.)

Penfield and Jasper (1954) report that similar turns can be associated with mid-frontal epileptogenic foci, and according to Gastaut (1954), they can accompany temporal epileptogenic foci.

CASE VI.—This was a case of involved epileptic giddiness. For three years, a woman patient, aged 26 years, had suffered at four to six-week intervals, from "fits", during which she had a spinning sensation in her head. There was, however, no loss of consciousness. There was no history of major seizures.

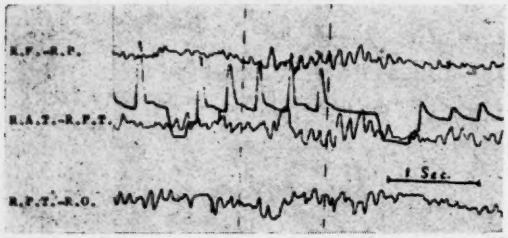

FIGURE V.
Right parieto-temporo-occipital epileptogenic focus. Key as for Figure III.

The electroencephalographic tracings showed spikes, some followed by a slow wave, in the right posterior temporal region. There were also a few slow-wave seizure patterns. Treatment with "Dilantin" and phenobarbital improved the condition and reduced the electroencephalographic spikes and seizure patterns. (See Figure VII.)

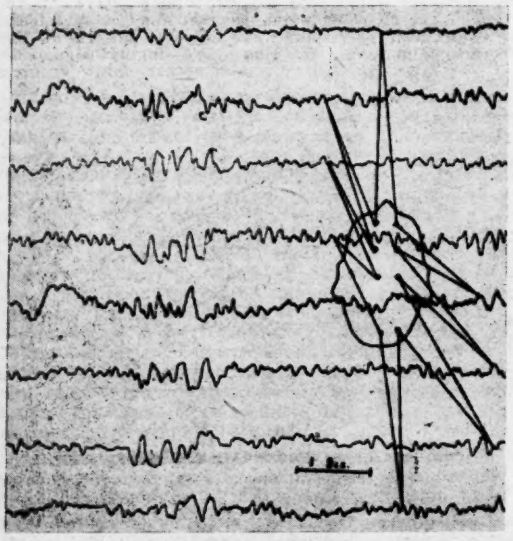

FIGURE VI.
Seizure pattern with spike in left frontal region.

***Epileptic Lapses with Clouded Consciousness.—Petit-mal* seizures associated with lapses of consciousness, clouded consciousness or loss of consciousness are phenomena with which most general practitioners and psychiatrists are familiar. Epileptic children suffering from frequent *petit-mal* seizures are listless and may appear to be feeble-minded.**

CASE VII.—This was a case of pyknolepsy in an apparently feeble-minded girl, aged four years. She was backward, could not talk and had a low frustration tolerance. The saliva dribbled from her mouth. The electroencephalographic tracings showed frequent high-voltage spike-and-wave seizure patterns, suggestive of frequent *petit-mal* lapses. The effect of treatment with "Diamox" was dramatic. Her attention and scholastic achievements increased considerably and the dribbling ceased. (See Figures VIII and IX.)

Pyknolepsy is a disease with an explosive onset at the age of about four years, lasting to about the age of 12 years, when it ceases spontaneously. The illness consists of frequent short, slightly monotonous *petit-mal* lapses, which recur almost daily for weeks, months and years (Hill and Parr, 1950). Usually the illness is detected only by electroencephalographic examination.

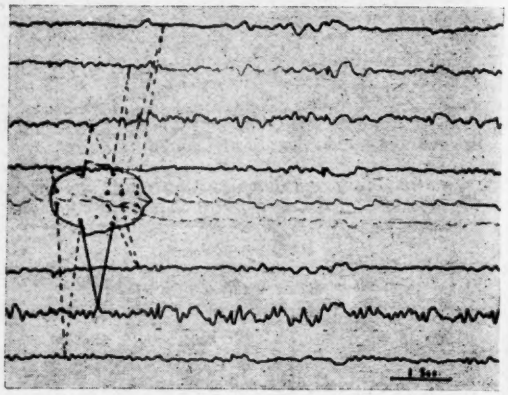

FIGURE VII.
Epileptogenic focus in right temporal region.

Migraine and Epilepsy.—About every second subject suffering from migraine has seizure patterns or focal abnormalities in his electroencephalogram (Dow and Whitty, 1947; Panzani and Boyer, 1955). In such subjects anti-convulsive drugs often relieve the headache (Heyck and Hess, 1955) and the injection of ergometrine causes the seizure patterns in the electroencephalogram to disappear (Hill and Parr, 1950). One sufferer from migraine, examined in this electroencephalographic department, not only illustrated this relationship in the electroencephalogram, but actually produced a clinical psychomotor seizure during hyperventilation.

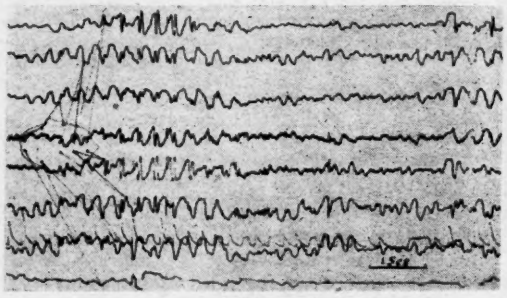

FIGURE VIII.
Seizure patterns in pyknolepsy.

CASE VIII.—This case was one of a youth, aged 17 years, of normal intelligence, who had suffered from migraine from the age of eight years. During the attacks of headache his hands became numb and he had falls, after which the colour of his face was ashen-grey. There was no history of major seizures. After taking 50 deep breaths during the electroencephalographic recording he became distressed, moved his

legs, rolled his body sideways and breathed more deeply still. The electroencephalographic tracings taken during that episode showed slow-wave seizure patterns. After ceasing to hyperventilate he became aware of his surroundings. He related that he had been unable to open his mouth, that he had been frightened and that he had never felt like that before. When he was questioned more closely, it became obvious that he had no clear idea of what had happened. (See Figure X.)

Organic Cerebral Abnormality Directly Responsible for Psychological Manifestations.

The electroencephalographic examination of psychiatric patients occasionally reveals organic abnormality which has previously been missed. In other patients, who were

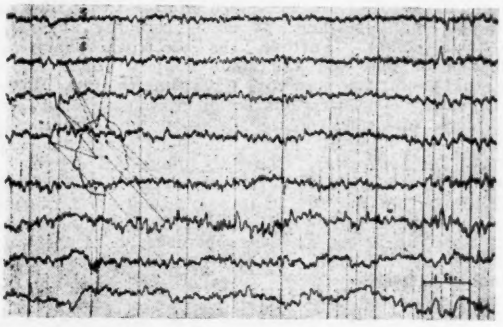

FIGURE IX.
Pyknolepsy treated with "Diamox".

suspected of some organic cerebral pathological condition, the electroencephalographic tracings were helpful in suggesting other special diagnostic procedures.

Organic Cerebral Abnormality Responsible for Mental Confusion.—Confusional states and coma can be caused by varous metabolic illnesses and by organic cerebral pathological conditions which may be related to psychiatric problems.

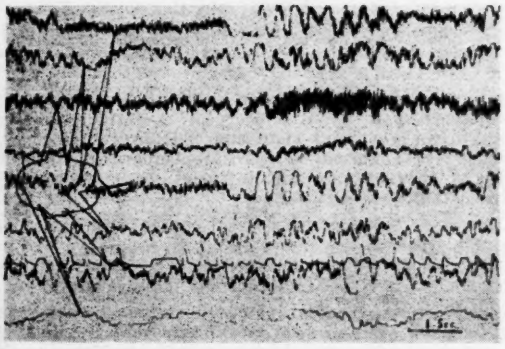

FIGURE X.
Seizure pattern in subject suffering from migraine.

CASE IX.—In this case a brain tumour was associated with mental confusion in a woman, aged 45 years, who until four months prior to her admission to a mental hospital had been quite well. She suffered from confusion, emotional flatness, gross retardation, blocking of thought and incontinence of urine and fæces. Later, she began to lose the power and use of her legs and developed mild hemiparesis. Bilateral papilloedema was eventually found. The electroencephalographic tracings showed continuous high-voltage slow-wave activity in both frontal regions, suggestive of a frontal space-occupying lesion. During a subsequent operation a glioma was found in the deep posterior part of the left frontal lobe. (See Figure XI.)

CASE X.—This was a case of general paralysis of the insane in a male migrant, aged 46 years, whose knowledge of English was poor, and who was diagnosed as suffering from paranoid schizophrenia. His blood did not react to the Wassermann test. Two years later he unexpectedly suffered a major seizure. The electroencephalographic tracings showed marked slow activity in both frontal regions, and to a lesser extent in other regions. After the injection of large amounts of "Megimide", the electroencephalographic tracings did not show any abnormality suggestive of epilepsy. Further Wassermann tests of both the blood and the cerebro-spinal fluid gave strongly positive results. (See Figure XII.)

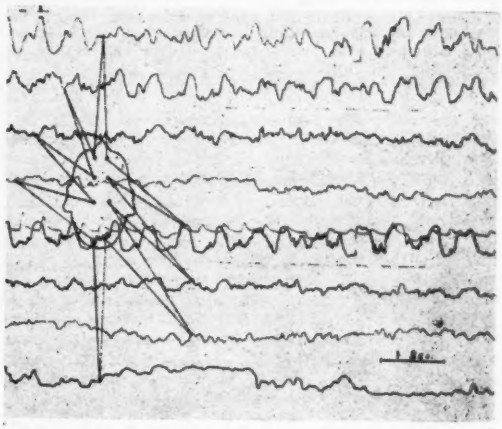

FIGURE XI.
Left frontal brain tumour.

In this case the electroencephalographic examination was instrumental in suggesting further investigations which led to the previously missed diagnosis of general paralysis of the insane.

CASE XI.—In this case, cerebral anoxia was responsible for mental confusion. A woman, aged 62 years, was knocked down by a motor-cycle. As a result of the accident she suffered from extensive chest injuries with multiple rib

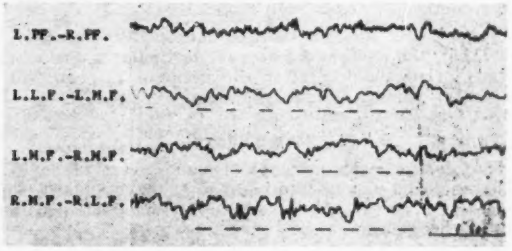

FIGURE XII.
General paralysis of the insane. Key as for Figure III.

fractures. There was no history of head injury or loss of consciousness. Soon after the accident her ribs were set under general anæsthesia. After this, she suffered from mental retardation, disorientation, confusion and insomnia. The electroencephalographic tracings showed considerable slow-wave activity in all regions, which became more marked during hyperventilation, and suggested a subacute diffuse cerebral disorder. The suggested diagnosis of the neurologist of cerebral damage due to anoxia during the anæsthesia was supported by the results of electroencephalographic examination. (See Figure XIII.)

Organic Cerebral Abnormality Responsible for Psychopathological Manifestations other than Confusion.—Occasionally a pathological condition may be localized and affect one or a few of the psychological functions only.

At least four such cases were discovered among the 1000 psychiatric patients examined in the electroencephalographic department at Mont Park. One illustrative case is presented here.

CASE XII.—This case illustrates lack of initiative as the probable result of partial accidental prefrontal lobotomy. A male patient, aged 51 years, exhibited such a degree of flatness of feeling and expression that the provisional early diagnosis of simple schizophrenia was made. Thirteen years previously, he had been involved in a motor-car accident; his skull was fractured and he was unconscious for 38 days, after which he lost all initiative and depth of feeling and was satisfied to drift along. With the exception of strabismus there were no neurological signs. However, his memory had suffered slightly, he began to suffer from a speech defect, he complained of numbness of his left leg and severe headaches during the night, his vocabulary became restricted and he made up neologisms. Occasionally it was observed

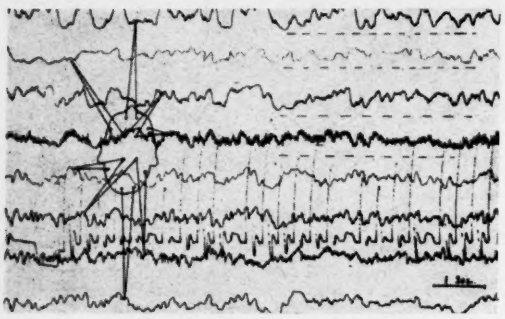

FIGURE XIII.
After-effect of cerebral anoxia.

that the right side of the face and left limbs were twitching. Lately, he had exhibited episodes of violence. The electroencephalographic tracings showed abnormal, low-voltage, theta-wave activity in both prefrontal and lateral frontal regions. Eight millilitres of "Megimide" given intravenously produced bursts of high-voltage fast waves and theta waves in the left prefrontal and left lateral frontal regions. The electroencephalographic abnormalities suggested organic damage in both prefrontal and lateral frontal lobes, and a minor epileptogenic focus in the left prefrontal and left mid-frontal lobe. (See Figure XIV.)

In this case, the electroencephalographic tracings, the twitching, the speech defect and particularly the marked change of personality after the accident all point to an accidental partial frontal lobotomy.

Methodology.

The electroencephalographic tracings were taken by an "Ediswan" electroencephalograph under standard conditions, and a "Walter" wave analyser was used as a routine procedure.

The customary methods of activation, such as hyperventilation, photic stimulation, acoustic stimulation, sleep and "Megimide" injection were applied when necessary. Bipolar recordings were taken as a rule, but monopolar tracings were also taken occasionally, when the voltages of various scalp areas had to be compared.

As has already been stated, the subjects investigated were 1000 psychiatric patients, the majority referred through various psychiatric out-patient clinics and the others through mental hospitals in the state of Victoria.

About one-third of the patients were non-cooperative, so that several methods of tranquillization had to be applied. These consisted of an explanation of the scope of the electroencephalographic procedure, the administration of chlorpromazine—which incidentally has a mild epileptogenic effect (Fabisch, 1957; Lyberi, 1956; Towler, 1957)—and, if this failed, of "Seconal" (Gibbs, 1952).

The clinical history sheet of every patient was available during the electroencephalographic recording, and all revelant items referring to the illness were noted.

Electroencephalographic Patterns in Psychiatry.

The interpretation of the electroencephalographic pattern, in terms of psychiatry, is sometimes unreliable and rather complex.

To begin with, correlation of psychiatric with electroencephalographic data is not always reliably consistent, but allows for some random variation. Some subjects with neurologically established cerebral abnormality have normal electroencephalographic patterns. About 10% to 15% of the general population have electroencephalographic patterns that deviate from the average electroencephalographic tracings to such an extent that they could also be manifestations of some cerebral abnormality (Schwab, 1951). Further, the electroencephalographic tracings of one and the same subject under different external conditions may vary from time to time. Different blood-sugar concentrations, temporary emotional states, artefacts, variations of the electroencephalographic technique, etc., may all affect the electroencephalographic pattern in some way. Clinically diagnosed cerebral disorders may not show up in the first electroencephalographic recordings, but may do so in the second or third recording, or may never do so at all. The same abnormality may show up in one electroencephalograph as one kind of pattern and in another electroencephalograph as a different kind of pattern.

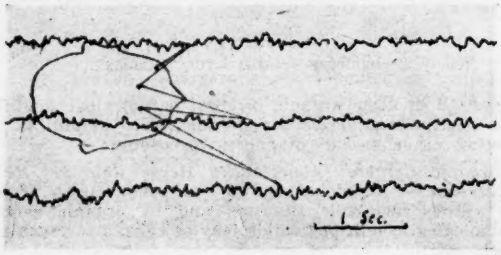

FIGURE XIV.
The low voltage theta waves are not very distinct in the primary tracings, but are convincingly demonstrated in the wave analyser tracings (not shown here).

It is easy to understand that strikingly abnormal electroencephalographic patterns of great magnitude are likely to have an abnormal significance. However, slightly or moderately abnormal electroencephalographic patterns, being so dependent on random variations, may or may not have pathological significance.

However, the confidence with which electroencephalographic patterns are interpreted relies mainly on already existing correlations emanating from many reputable neurological and electroencephalographic laboratories.

The electroencephalographic pattern has to be correlated with several other sets of data: (i) psychiatric data; (ii) neurological data; (iii) history or other evidence of brain injury; (iv) history or other evidence of vascular, inflammatory or metabolic illnesses likely to affect the brain; (v) effect of drugs; (vi) an epileptic seizure during the electroencephalographic recording. If the data belonging to the various sets fit into the concept of a single cerebral abnormality, the argument for the interpretation of an electroencephalographic pattern is strengthened. Good correlations and correctly significant interpretations of the electroencephalographic tracings are obtained if one of these sets of data is varied or manipulated, and if this procedure results in a corresponding variation of the electroencephalographic pattern.

For instance, the administration of anti-convulsant drugs may change the electroencephalographic pattern as well

as supposed epileptic behaviour, and in this case the changes of the two sets of data may allow the inference of a correlation between them. The best correlation of the electroencephalographic pattern with any form of epilepsy is obtained if the onset and end of a clinical seizure coincide with the onset and end of an electroencephalographic seizure pattern. Statistics of the percentage of abnormal electroencephalographic tracings in epileptics and sufferers from brain tumours have been published and are of practical help. However, in many cases so many aspects have to be considered, and the random factors are of such major dimensions, that a computation of the statistical probability becomes impracticable.

Hence, much of the clinical evaluation depends on the experience, judgement and systematic investigation procedures of the individual electroencephalographer.

Ætiology.

The statistics of Gibbs and Gibbs (1952) suggest that for the majority of cases of epilepsy, the ætiology could not be determined. However, they found that, depending on the type of epilepsy, 4·5% to 9% of epileptics had a family history of epilepsy; 3% to 12·5% had a history of birth injury; 3% to 8% had a history of encephalitis; 1·2% to 2·2% of psychomotor epileptics had a history of cerebro-vascular disease. In a comparatively small proportion of cases of epilepsy there was a variety of ætiologies, such as meningitis, cerebral space-occupying lesions, etc.

In this electroencephalographic department, it was found that, out of 185 frank epileptics, 72 (39%) had a history associated with clinical conditions which could have been responsible for their illness. Eighteen (10%) of the epileptics had a family history of epilepsy; 49 (27%) had a history of birth injury; and five (3%), had a history of encephalitis. These figures are suggestive, though not a valid indication, of the ætiology. There are various reasons for a strong bias underlying these figures. A history of cerebral trauma, birth injury or encephalitis in epileptics may be a mere coincidence, and does not necessarily signify that the cerebral abnormality was the cause of the epilepsy. Brain-damaged epileptics were selected for electroencephalographic investigation in preference to those without a history of brain damage. Only mentally ill epileptics were examined.

Nevertheless, the figures of Gibbs and Gibbs and those of this electroencephalographic department suggest that the ætiological factors responsible for epilepsy were mainly birth injury, cerebral trauma, heredity and encephalitis.

It is not generally realized that various physiopathological conditions may trigger latent epilepsy or aggravate open epilepsy (Gastaut, 1954). Such temporary triggering factors are alcoholic bouts, massive ingestion of liquids, physical fatigue, emotional stress (particularly in children), hypoglycæmia, drowsiness and constipation.

Indications for Electroencephalographic Examination.

When all relevant factors are taken into account, the following indications for electroencephalographic examination of psychiatric patients suspected to suffer from cerebral disorder with psychological manifestations, have been found to be useful.

1. Frank epilepsy associated with paroxysmal psychological disturbances, particularly if there are also neurological abnormalities, a history of head injury, birth injury, encephalitis, other cerebral disorder or backwardness in children.

2. Paroxysmal psychological disturbances without frank epilepsy, particularly if associated with convulsions in childhood, neurological abnormalities, a history of head injury, birth injury, encephalitis or other cerebral disorder, alcoholism, or backwardness in children.

3. Intellectual deterioration, confusion or memory defects, particularly if associated with neurological abnormalities, or metabolic diseases, vascular diseases, alcoholism, suspicion of cerebral abnormality or history of head injury.

4. History of head injury or suspicion of cerebral abnormality in a mentally ill patient.

5. Any badly defined mental illness with manifestations which, put together, cannot be integrated to form the pattern of a known functional psychosis.

Summary.

1. The electroencephalographic tracings of 1000 psychiatric patients were reviewed in the electroencephalographic department at Mont Park.

2. Dissatisfaction was expressed with the traditional approach of interpreting the electroencephalographic tracings in neurological terms only. A consideration of psychopathological information in addition to electroencephalographic data is advocated in the investigation of psychiatric patients.

3. Of the 1000 psychiatric patients observed, 48% had abnormal electroencephalographic patterns, 15% had borderline patterns and 37% had patterns which were judged to lie within normal limits.

4. It was found that psychopathological manifestations as a result of cerebral abnormality, when none had been suspected previously, were more common (over 1·7%) in the psychatric population than the general practitioner or the psychiatrist seems to realize. The most common epileptic psychological aberrations were: furors; "black-outs" associated with abnormal behaviour; lack of attention; apparent feeble-mindedness; abdominal pain; giddiness; migraine; hallucinations and dreamlike states associated with feelings of unreality.

5. Electroencephalography was found the be helpful in discovering, and sometimes in confirming, the clinical diagnosis of cerebral abnormality responsible for psychopathological manifestations. The most common organic cerebral conditions encountered were arteriosclerosis, cortical atrophy, brain injury, general paralysis of the insane and cerebral tumours. The most common psychopathological manifestations were confusion, memory defects, poor orientation, backwardness, lack of initiative and speech defects.

6. The criteria of evaluation of electroencephalographic tracings in psychiatry, in terms of reliability and significance, are discussed, and the indications for electroencephalographic examination of psychiatric patients are formulated.

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MATERNAL AND CHILD WELFARE: THE ROLE OF THE GENERAL PRACTITIONER.[1]

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Mile End, South Australia.

GENERAL PRACTICE is family medical practice, and a substantial part of the general practitioner's work is based upon the mother-child relationship, both in sickness and in health. One of the ideals of good general practice should be the preservation of health in mother and child, and in the last fifty years, which have seen so many changes, one of the most significant changes in family medical practice has been the development of a tradition of well-child care, and of efficient ante-natal and post-natal care.

The tradition of visits to the doctor in health rather than in sickness is growing; and we may say that medical practice will approach perfection when the people who visit the family doctor all go to seek advice or treatment that will maintain their good health—when family doctors become, in effect, practitioners of preventive medicine.

General practice is the front line in the war between man and disease. In this front line there is an enlarging salient that reaches out to and sometimes crosses the frontier between medicine and sociology. If medicine is the study of the anatomy, physiology and pathology of the human individual, sociology is the study of the anatomy, physiology and pathology of human groups. It is becoming increasingly important for doctors, especially general practitioners, to have some comprehension of this science of sociology. This is very obvious when we consider a topic like maternal and child welfare; more and more the family doctor's task has sociological overtones.

What is the present pattern of family life in Australia? The pattern is the same in most countries of the western civilized world that enjoy a comparably high standard of living. Our society is becoming increasingly urbanized, increasingly sophisticated, increasingly preoccupied with material possessions—a modern home, a car, a refrigerator, and so on, all very often being bought on time payment.

The average suburban mother is in her late teens or her early twenties when she has her first baby. She will usually go on to have two, three, or occasionally four children, judiciously spaced over rather less than ten of her thirty odd years of reproductive life. She has no home help, though she usually has a mother or a mother-in-law who can rally around at times of stress. Alternatively, this young mother may still be living in one or two rooms in her parents' home, in which case she will very likely go out to work, leaving her offspring in charge of grandparents. In any case she will often go out to work as soon as her children are old enough to be disposed of for the necessary few hours during the day. Of the social pressures that oblige her to do this, the greatest is the growth of hire-purchase economy, with its demands on the family budget. It also seems to have become fashionable to some extent for young married women to have a job, although this situation could be changed by legislation relating to the payment of women in industry. These generalizations cover a good many of the young families living in metropolitan Australia in mid-century. Both parents are physically pretty healthy, and so, by and large, are the children.

It must be added that there is an increasing dilution of this metropolitan population by families from entirely different cultural and traditional backgrounds—the European immigrants in our community. Some of these people came from what are basically peasant societies in places like southern Italy, Greece, and the Balkan peninsula, and they have standards of hygiene and nutrition that are different from those that we accept as ideal.

This is a very simplified word picture of our way of life. We may not necessarily approve of all its features, but it is not for us to pass judgement upon it. We must ask now, in respect of maternal and child welfare, are the needs of this society being met?

Some of its needs have been very well met, as we can infer by comparing our tables of infant mortality and the like, either with corresponding tables from fifty years ago, or with figures from such countries as Mexico or Egypt (Brockington, 1958). Obviously there is no cause for complacency about our relatively low infant mortality rate, which has not yet reached an irreducible minimum; nor will there be cause for complacency when babies no longer die of neonatal sepsis or of gastro-enteritis. However, it is appropriate for us to turn from a consideration of mortality reduction to the related question of the reduction of morbidity.

I should like to consider three topics in particular that bear on this question of the reduction of morbidity: infant nutrition, ante-natal and post-natal care and the mental health of mother and child.

Infant Nutrition.

It has been suggested that the giving of detailed advice on infant feeding may be an obsolete or at any rate an obsolescent discipline.

Our preoccupation with the virtue of obesity in infancy may be what has led to the premature caking up of our coronary arteries (as has been suggested, for example, by "Richard Cobbleigh", one of the pseudonymous contributors to the "Widdicombe File" in *The Lancet*, 1956). I should like to hear what a pædiatric pathologist has to say about the frequency with which atheromatous changes are seen in young children. Perhaps we may find it necessary before long to reappraise some of our ideas on infant feeding.

While there are some young mothers who undoubtedly need detailed advice on the correct way to feed their babies, encouragement to persevere with breast feeding, and advice about the preparation of uncontaminated artificial feeds, this may no longer be the primary need of a good many young mothers who attend infant welfare clinics, such as those conducted in South Australia by the Mothers' and Babies' Health Association.

I can recall only about two or three frankly malnourished babies that I have seen in five years of suburban general practice. It is not common even to be consulted by mothers seeking detailed advice about infant feeding. In 1958, I recorded this only once as the primary reason for consultation—though, of course, I saw perhaps two or three dozen girls to whom I gave advice in the course of routine post-natal consultations, or when they brought their babies in for their inoculations. Most mothers seem to know that they can get the advice they need about infant feeding from a mothercraft-trained nurse at the "Mothers' and Babies'" clinic. The criticism that this advice may be confusing to an inexperienced mother is ill founded. The giving and taking of advice are like the seed and the soil of the parable. Sometimes the seed falls on the stony ground of inattention, or among the thorns of prejudiced or preconceived ideas. Alternatively, the seed of good advice may be encased in such a tough husk of verbiage that it cannot germinate, even in the most receptive soil of an intelligent young mother's mind. I have seen this happen even when the seed has been disseminated by an eminent consultant.

In my practice, one patient in every four or five is a "New Australian" immigrant from the continent of Europe. I have been impressed by the numbers of these people I see, especially those of peasant extraction, who obviously make use of the facilities of the "Mothers' and Babies'" clinics. It is very good to see this; in spite of the influence of the black-clad matriarch hovering in the background and, no doubt, grumbling in Greek, Italian or Serbo-Croatian that it is flying in the face of nature, a good many of these girls, to judge by the homes I go into, do boil their milk and their bottles, and do try to keep the flies and the older children away from the teats.

[1] *Read at a meeting of the South Australian Branch of the British Medical Association on July 30, 1959.*

When these girls bring their babies into the consulting rooms for their inoculations, they very often have the little pink or blue booklets issued by the Mothers' and Babies' Health Association, and as I record their inoculations, I notice also the carefully kept record of weight and feeding that means regular attendance at the clinic. Not all are like this, of course, but a significant number seem to be; and the numbers could doubtless be increased by appropriate publicity — for example, in the Italian language newspaper *La Fiamma*.

These observations are in accord with the findings of a team of social anthropologists under the leadership of Margaret Mead (1955). By means of carefully conducted field studies in Greece, Burma, the Tiv of Nigeria, the Palau Islands and the Spanish America of New Mexico, these workers showed how and in what ways the standards of peasant peoples are raised towards the levels of a technologically more advanced culture. I do not anticipate that our good record of low infant mortality will be upset by the influx of these people from peasant societies.

Ante-Natal and Post-Natal Care.

In 1949, a Joint Committee of the Royal College of Obstetricians and Gynæcologists, the Population Investigation Committee, and the Institute of Child Health published their "Population Studies", which showed, among many other interesting things, that in Great Britain 40% of mothers suffered some discomfort or disability attributable to the effects of pregnancy and labour in the two years after childbirth. In 26% this disability becomes permanent. Professor Leslie Banks of Cambridge has pointed out (1953) that a very great deal of this disability is preventible. The position is probably very similar in Australia.

An effective programme of ante-natal and post-natal exercises may do much to prevent the development of some of the distressing gynæcological and orthopædic disorders that may afflict the girl who has borne children. The need for these exercises, especially continued post-natal exercises, becomes greater with each successive pregnancy, with each successive period of strain on abdominal, spinal and pelvic muscles.

At maternal and child welfare centres, such as the Mothers' and Babies' Health Association Centres in South Australia, it may become possible in the future to conduct classes for expectant mothers on a wider scale than at present. If the classes included some elementary instruction in the basic anatomy and physiology of pregnancy and labour, as well as information about the hygiene of pregnancy and preparation of the mother for breast feeding, both physically and psychologically, so much the better. At these classes, a girl who may feel lonely, withdrawn, perhaps not a little afraid in her first pregnancy, meets others in the same state as herself, and so experiences the reassurance that comes from belonging to a group. This alone almost justifies the existence of such classes, whatever may be the merits of ante-natal exercises and relaxation.

There are many practical difficulties that militate against the organization of classes for post-natal exercises; but it should be possible for family doctor, obstetrician and mothercraft-trained nurse to stress to mothers the desirability of continuing post-natal exercises over a prolonged period of time. Apart from any other consideration, a mother who is preoccupied by ill-health cannot devote to her children all the care they need if they are to be given a good start in life.

The Mental Health of Mother and Child.

The pattern of illness in the community is changing remarkably during the lifetime of those of us now in practice. More and more the dominant diseases have causes that appear to be directly related to changing social *mores*, rather than to microbes present in the environment. Two diseases in particular are relevant to this present discussion—child delinquency and psychoneurosis. The causative factors of these two disorders are extremely complex, and are by no means fully understood. Both may be symptomatic of a fundamental disorder in our civilization; a study of the history and literature of past civilizations, such as the Greek and Roman civilizations, rather suggests that this is so.

In our over-urbanized society, the individual may seek to preserve his individuality by erecting about his personality a barrier to cut himself off from his fellow men. This is how he remains unique, an island entire unto himself. But the human animal is by nature grègarious, and it seems that his psyche may suffer if he attempts to remain aloof from his fellows in this or other ways. Perhaps there are many young wives and mothers obliged to live within the restraining privacies of a city, when their instincts cry out for the companionship of the village, who may thus become maladjusted, and so psychoneurotic. As social meeting places, maternal and child welfare clinics such as those of the Mothers' and Babies' Health Association may thus fulfil a valuable social function. It is possible that much more could be done towards better community mental health by appropriate encouragement of this and other similar organizations to play a greater part in group psychotherapeutic activities, such as the rehabilitation of people who have been emotionally or mentally disturbed.

At the same time, as the American sociologist David Riesman and his co-workers have pointed out (1950), all manner of seductive influences are at work to undermine the stability of the mother-child relationship; the economic need for the mother to work, and the pressure of synthetic forms of entertainment such as films, television and so on, are only two of the more obvious ways in which a mother may be distracted from her prime duty to her child.

Dr. John Bowlby, in a study carried out for the World Health Organization (1953), has shown conclusively that maternal care in infancy and early childhood is essential for the preservation of mental health. To quote from his report:

> This is a discovery of which the importance may be compared to that of the role of the vitamins in physical health, and is of far-reaching significance for the prevention of mental ill-health.

It is strongly suggested that, if we are to eradicate child delinquency from our community, our preventive measures must start at the very beginning, at the birth of the baby—perhaps even before. Very possibly the same may be true of much psychoneurosis.

The last half-century has seen the fulfilment of many of our hopes for a physically healthy community. Perhaps the next half-century will see the firm establishment of sound mental health in the community. This is going to be a very difficult task indeed; but if it depends upon any one factor more than all others, then that one must surely be the integrity of the family as the fundamental unit of society.

In this highly mechanized and increasingly complex civilization, there are many strong social forces operating together, all tending to disrupt family unity, with very widespread consequences, of which the examples of impaired mental health that we see with increasing frequency are perhaps only a relatively minor symptom. In my more pessimistic moments I sometimes wonder whether man is lacking altogether in the wisdom needed to live successfully with all his recent inventions. Just to take one very simple example, the man who lives in a dormitory suburb more than an hour's travelling time from his work may have only fleeting glimpses of his wife and children for five days out of seven, and very often the force of prevailing social custom may even conspire to keep the family apart at week-ends. Such a family may cease to exist as a unit, with each member aimlessly going his own way. If the housewife and mother also goes out to work, the tendency for this to occur is even greater. The real trouble begins when a child of such a family is in need of emotional or psychological guidance, which is not readily forthcoming from a group

of individuals who have, to a certain extent, lost their easy intimacy with one another.

Very few people in the community are in a position to exercise any favourable influence upon individual families that are in danger of physical or psychological disruption in this or other ways. Family doctors are among the few people who ought to be in such a position—that is, if such a "family" has not so completely lost its unity that it no longer even has one doctor, but several. However, almost all family doctors lack the necessary training, and many lack the interest to deal with problems of this nature—problems that have no appearance of urgency, and that may be so abstract that it is hard even to define their extent. I have the feeling very strongly that this is one way in which medical thought and medical education have lagged a long way behind the needs of the community.

Conclusion.

I have said nothing about several other aspects of maternal and child welfare that are of great importance: the problems of handicapped children; the problems of children deprived of parents, home, security; the problems of neglected and ill-treated children, and of illegitimate children. Accident prevention is becoming an increasingly important problem in this age of increasing domination of machines over men. Family planning, by means of some form of birth control, is a subject whose far-reaching implications make it necessary to omit it altogether from this discussion, simply for lack of time.

If I must summarize these remarks, I would restate my main theme this way. The family, the fundamental unit of society, is all-important as a stabilizing force. The family is being subjected to very strong disruptive pressures, which must be resisted if our society is not to be seriously damaged. The medical profession and its ancillary services should be in a position to resist these disruptive pressures; but almost all the basic knowledge needed to do this is lacking, and the little knowledge we have plays no part in formal medical education.

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HEPATO-SPLENOMEGALY ASSOCIATED WITH ANÆMIA IN NEW GUINEA NATIVES.

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ENLARGEMENT of the liver and spleen is seen and treated frequently amongst the indigenous people of tropical countries. The causes of this syndrome differ in different countries. Hepato-splenomegaly, as it occurs amongst the natives of New Guinea, was investigated and described by Black (1954). Malaria was mentioned as the probable causative factor. Fawdry (1955) described a syndrome of hepato-splenomegaly and anæmia amongst the Arabs in the Aden Protectorate. Jansen (1959), describing a similar syndrome in Netherlands New Guinea, writes:

> There seems to be a correlation between enlargement of the liver and the hæmoglobin level: Those with a distinctly palpable liver have lower hæmoglobin values than others with an impalpable liver. This is most marked on Biak, which has the highest malaria endemicity of all places visited; the difference in the Mappia District, the only place without malaria, is only small.

A similar syndrome is present amongst the natives of the Territory of Papua and New Guinea, and the purpose of this paper is to record some observations made during the last five years of clinical and field survey work. Large-scale surveys were carried out in Kieta Sub-District, on Bougainville Island, in the Goilala Sub-District of Papua and in the Goroka Sub-District of the Eastern Highlands District, New Guinea. These were initially made as blood-group surveys (Kariks, Kooptzoff and Walsh, 1957; Kariks, Kooptzoff, Cotter and Walsh, 1958), and revealed also an interesting side-light on the hæmoglobin values of the natives of Bougainville (Cotter, Kariks and Walsh, 1958).

TABLE I.
Persons Examined.

Subjects.	Adults.		Children.	Total.
	Male.	Female.		
Bougainville village natives	497	510	413	1420
Kieta hospital patients ..	11	11	17	39
Goilala village natives ..	305	100	18	423
Goroka village natives ..	349	303	890	1542
Total	1162	924	1338	3424

TABLE II.
Enlarged Spleen and Liver.

Subjects.	Adults.		Children.	Total.
	Male.	Female.		
Bougainville village natives	16	49	72	137
Kieta hospital patients ..	10	10	17	37
Goilala village natives ..	0	1	0	1
Goroka village natives ..	1	0	35	36
Total	27	60	124	211

TABLE III.
Enlarged Liver Only.

Subjects.	Adults.		Children.	Total.
	Male.	Female.		
Bougainville village natives	3	10	5	18
Kieta hospital patients ..	1	1	0	2
Goilala village natives ..	1	0	0	1
Goroka village natives ..	2	10	217	329
Total	7	21	222	250

Further study of this survey material, a special survey in Goroka Sub-District and some clinical observations made in different hospitals during the years 1954 to 1959 revealed another sidelight on some factors which, although frequently observed, had been seldom described. These observations may be of some interest and may lead to further investigations.

Materials and Methods.

Blood was collected and the hæmoglobin was estimated by the method described by Cotter *et alii* (1958). Hæmoglobin estimation amongst the separate group of infants and children of the Goroka Sub-District was carried out by the Sahli method upon blood obtained from the fingertip or the heel by skin puncture. Before collection of the blood, each individual was physically examined, and special attention was paid to the presence of liver and spleen enlargement. This examination was carried out with the subject in a recumbent position on a canvas camping bed.

TABLE IV.

Enlarged Spleen Only.

Subjects.	Adults.		Children.	Total.
	Male.	Female.		
Bougainville village natives	114	127	151	392
Kieta hospital patients ..	0	0	0	0
*Goilala village natives ..	9	12	2	23
Goroka village natives ..	0	3	0	3
Total	123	142	153	418

TABLE V.

Anæmia[1] as it Affected People in Different Groups Shown in Tables II to IV.

Subjects.	Spleen and Liver Enlargement.				Total.
	Spleen and Liver.	Liver Only.	Spleen Only.	None.	
Bougainville village natives:					
Men	9	0	10	18	37
Women ..	9	1	16	27	53
Children ..	30	1	27	23	81
Total ..	48	2	53	68	171
Kieta hospital patients:					
Men	10	1	0	0	11
Women ..	10	1	0	0	11
Children ..	17	0	0	0	17
Total ..	37	2	0	0	39
Goilala village natives:					
Men	0	0	0	1	1
Women ..	0	0	0	1	1
Children ..	0	0	0	1	1
Total ..	0	0	0	3	3
Goroka village natives:					
Men	0	0	0	0	0
Women ..	0	0	0	1	1
Children ..	35	217	0	109	361
Total ..	35	217	0	110	362
Grand total	120	221	53	181	575

[1] Hæmoglobin value 10·0 grammes per 100 ml. and less.

Splenic enlargement was recorded as recommended by Hackett. Liver enlargement was recorded by counting the number of fingers' breadth by which the liver was enlarged below the right costal margin in the mid-clavicular line. There are many objections relating to the accuracy and scientific value of this method. However, it is invaluable in primitive field conditions and saves much time. The liver was recorded as enlarged when its edge was palpable at least one finger's breadth below the costal margin in the mid-clavicular line with the lung in expiration.

A person was classified as "anæmic" when the hæmoglobin value was 10·0 grammes per 100 ml. or less. Persons who had hæmoglobin values between 10·1 and 13·0 grammes per 100 ml. were classified as "intermediate". Those having a hæmoglobin value of 13·1 grammes per 100 ml. and over were classified as "normal". The intermediate classification is arbitrary and bears some significance for the purpose of this survey only, because it was difficult to decide at what level to classify a person as anæmic, since different authorities regard different values as normal.

TABLE VI.

Intermediate Group[1] in Relation to Groups Shown in Tables II to IV.

Subjects.	Spleen and Liver Enlargement.				Total.
	Spleen and Liver.	Liver Only.	Spleen Only.	None.	
Bougainville village natives:					
Men	6	1	60	117	184
Women ..	29	5	79	193	306
Children ..	35	4	108	125	272
*Total ..	70	10	247	435	762
Goilala village natives:					
Men	0	0	2	9	11
Women ..	0	0	4	23	27
Children ..	0	0	1	1	2
Total ..	0	0	7	33	40
Goroka village natives:					
Men	0	0	0	2	2
Women ..	0	2	0	9	11
Children ..	5	0	0	507	512
Total ..	5	2	0	518	525
Grand total	75	12	254	986	1327

[1] Hæmoglobin values 10·1 to 13·0 grammes per 100 ml.

For the purpose of this paper, a total of 3424 persons were included on whom all the relevant examinations were made, although more than 6000 people were surveyed. In all, 1162 adult males, 934 females, and 1338 children of both sexes from birth to 15 years of age have been surveyed.

Results.

The results of this survey are shown in Tables I to VIII. Briefly summarized, they reveal the following facts.

Out of 3424 persons examined, 211 had an enlarged liver and spleen, 250 persons had an enlarged liver only, and 418 persons had an enlarged spleen only.

Five hundred and seventy-five persons were classified as "anæmic", as their hæmoglobin values were 10·0 grammes per ml. or less; 1327 persons, whose hæmoglobin values were between 10·1 and 13·0 grammes per 100 ml. were classified as "intermediate"; 1522 persons had normal hæmoglobin values (13·1 grammes per 100 ml. and over).

Out of 211 persons with hepato-splenomegaly 120 were anæmic, 75 were in the intermediate group and only 16 had normal hæmoglobin values.

Hepato-splenomegaly and anæmia were proportionally higher amongst children and females of child-bearing age.

Discussion.

Anæmia is widespread amongst the village population of New Guinea. It affected 575 persons, or 16·8% of the total population examined. The percentage rises steeply to 55·5 if the intermediate group is added to it. In practice, this means that every second native in New Guinea is suffering from anæmia or has a reduced hæmoglobin value. This bears some significance upon the country's economy and public health problems. This may also partly explain the mental lethargy and backwardness of the native people.

On Bougainville, where the climate is hot and humid and malaria is hyperendemic to holoendemic, anæmia was found to be more evenly distributed amongst the child and adult population, although proportionally the children were more frequently amongst the sufferers. In the Goroka Sub-District, where the climate is cooler owing to the higher altitude (5000 to 6000 feet), and where malaria is only sporadic, the infant and child population was mainly affected. On Bougainville, the anæmia was mainly of the microcytic iron-deficiency type, and responded favourably to iron and antimalarial drug therapy. Anæmia amongst the Goroka natives was of normocytic normochromic character, and the treatment required was a protein-rich diet, iron, vitamin B_{12} and folic acid.

TABLE VII.

Normal Hæmoglobin Values[1] in Different Groups Shown in Tables II to IV.

Subjects.	Spleen and Liver Enlargement.				Total.
	Spleen and Liver.	Liver Only.	Spleen Only.	None.	
Bougainville village natives:					
Men ..	2	1	49	224	276
Women ..	7	4	28	112	151
Children ..	5	0	15	40	60
Total ..	14	5	92	376	487
Goilala village natives:					
Men ..	0	1	8	284	293
Women ..	1	0	6	65	72
Children ..	0	0	2	13	15
Total ..	1	1	16	326	380
Goroka village natives:					
Men ..	1	3	0	343	347
Women ..	0	8	3	280	291
Children ..	0	0	0	27	27
Total ..	1	11	3	640	655
Grand total	16	17	111	1378	1522

[1] Hæmoglobin value 13·1 grammes per 100 ml. and over.

This survey revealed that there are two distinct causes responsible for anæmia in New Guinea. In the coastal areas, the principal cause is malaria. A small-scale survey was carried out amongst the natives of Arawa village on Bougainville. This village had a total population of 98. Thick and thin blood slides were examined from 72 persons; 33% of these had malaria parasites in their peripheral bloodstream. At the time when these slides were taken, none of the subjects complained of being ill.

Anæmia, as it was seen amongst the Goroka native infants and children, was a manifestation of malnutrition. A separate survey was carried out to determine the extent of malnutrition amongst the Goroka native children. A total of 1223 infants and children was examined. This survey revealed that of all the children examined, 13·3% were suffering from malnutrition. This percentage rises very steeply to 44·0 when only the most vulnerable age group—between one and three years—is included. Hepato-splenomegaly and anæmia, as seen amongst the adult population around Goroka, were frequently associated with malaria. Clinical hepato-splenomegaly and anæmia were seen in three patients with macroglobulinæmia in Goroka.

Foy and Kondi (1958) were of the opinon that liver and spleen enlargements had no direct correlation with anæmia. Jansen (1959), as quoted above, found that there was some correlation between these two clinical findings. The results of this survey, and the clinical observations shown in Table VIII indicate that there is some definite correlation between hepato-splenomegaly and anæmia. It is not clear yet which is the primary factor, and which are the secondary effects. It appears that there is a kind of vicious circle which perpetuates the condition if this is not interrupted by clinical treatment.

The following points of interest should be noted: (*a*) with the clinical relief of anæmia, the size of both liver and spleen diminishes (Table VIII); (*b*) the more severe the anæmia, the greater is the hepatic and splenic enlargement; (*c*) among 211 persons who had enlargement of liver and spleen, only 16 had normal hæmoglobin values.

The actual mechanism of this type of hepato-splenomegaly is still not clear. However, it is certain that no single factor, but a multiplicity of factors, are responsible for it. Ray (1954), as quoted by Jansen (1959), has found that monkeys inoculated with *Plasmodium cynomolgi* and kept on normal diet did not show any significant changes in the liver. Animals kept on protein-poor diets and inoculated with *P. cynomolgi* developed fatty infiltration of the liver.

Native diet is rather poor in proteins, and this is particularly so in relation to infants and small children, whose protein requirements are higher per kilogram of body weight than those of adults. This may explain the higher rate of hepato-splenomegaly amongst the Bougainville child population.

Bascoulergue (1957) in French Equatorial Africa, and Hsieh Hsieh Chen (1957) on Formosa, have observed that hepato-splenomegaly was frequently associated with malaria infection, and that children were more frequently affected. It was assumed, also, that hyperplasia of the reticulo-endothelial system occurred in the building up of malarial immunity, and both the liver and spleen are rich in this tissue.

In kwashiorkor, there is always fatty infiltration of the liver with consequent liver enlargement.

Anæmia, by itself and if prolonged, causes fatty infiltration of the liver cells, and this may account for liver enlargement in some cases.

A frequent clinical observation is that people suffering from severe anæmia have an enlarged heart, and they often die in congestive heart failure. The mechanism can be explained as follows. Prolonged anæmia causes fatty infiltration of the liver cells and fatty degeneration of the heart muscle. Fatty degeneration of the heart muscle, associated with continued physical activity, leads sooner or later to congestive heart failure with cardiac enlargement, hepatic congestion, and dependent œdema and ascites. Enlargement of the heart is a frequent observation amongst native individuals in the tropics (Jansen, 1959; Payet *et alii*, 1956). Post-mortem examinations of persons who have died in congestive heart failure, and who prior to their death had been suffering from anæmia with hepato-splenomegaly, revealed hepatic congestion with central necrosis and peripheral fatty infiltration of the liver, and also fatty degeneration of the heart muscle.

Histological examination of biopsy and necropsy specimens by different workers showed fatty infiltration (Waterlow, 1954) or central fibrosis, or central necrosis associated with fibrosis (Achar and Chacko, 1954).

Summary.

Hepato-splenomegaly associated with anæmia is a very frequent clinical and field observation in New Guinea.

A multiplicity of factors in different combinations—malaria, malnutrition, anæmia and congestive heart failure are thought to be responsible for this syndrome.

TABLE VIII.
Kieta Hospital Patients.

Patient's Number.	Sex.	Age in Years.	Liver Size on on Admission.[1]	Spleen Size on on Admission.[2]	Hæmoglobin Value on Admission.[3]	Clinical Diagnosis.	Liver Size on Discharge.[1]	Spleen Size on Discharge.[2]	Hæmoglobin Value on Discharge.[3]	Serum Protein Content.[4]	Remarks.
1	M.	4	2	3	7·5	Malaria, anæmia.	0	0	12·7	—	
2	M.	36	2	4	6·3	Anæmia.	1	2	12·3	—	
3	M.	18	2	3	9·0	Yaws, anæmia.	0	3	11·1	—	Absconded.
4	F.	3	2	2	7·5	Anæmia.	1	0	12·4	—	
5	F.	10	1	2	10·0	Yaws.	1	0	12·8	—	
6	M.	19	2	0	10·0	Lobar pneumonia.	Palpable	0	15·0	—	
7	F.	68	2	0	10·0	Bronchopneumonia.	Palpable	0	12·4	—	
8	M.	24	2	4	8·4	Anæmia.	1·5	4	10·8	—	
9	F.	36	2	4	9·9	Yaws.	1	3	11·1	—	Absconded.
10	M.	8	2	4	9·1	Tropical ulcer.	2	3	12·4	—	
11	F.	5	3	2	9·9	Anæmia, malaria.	1	0	12·0	—	
12	M.	16	2	5	7·8	Anæmia.	1	4	11·4	—	
13	M.	10	1	3	6·0	Anæmia, tropical ulcer.	0	3	10·8	—	
14	F.	28	3	3	9·3	Anæmia.	0	3	11·4	—	
15	F.	48	3	2	6·9	Anæmia, bronchitis.	1	2	10·8	—	
16	F.	6	3	2	7·5	Malaria, anæmia.	0	1	12·0	—	
17	M.	56	3	3	9·6	Bronchopneumonia.	1	2	11·2	—	
18	F.	45	3	4	7·5	Bronchitis, anæmia.	2	3	11·2	—	
19	F.	58	3	3	4·5	Bronchopneumonia.	1	2	11·3	—	
20	M.	10	2	3	7·5	Tropical ulcer.	2	1	10·8	—	
21	F.	25	2	2	3·3	Anæmia.	1	1	10·8	—	
22	F.	36	3	4	3·9	Anæmia.	1	2	10·5	—	Abortion at five months.
23	F.	25	2	2	6·0	Anæmia.	1	1	11·0	—	
24	F.	3	2	2	2·4	Anæmia.	1	1	12·4	8·5	
25	F.	3	2	2	4·2	Anæmia.	1	0	10·8	7·8	
26	F.	19	5	6	4·5	Hepatitis.	—	—	—	7·9	Died.
27	M.	29	2	4	8·7	Malaria.	0	3	12·0	8·9	
28	F.	4	1	1	7·5	Anæmia.	0	2	12·3	7·2	
29	F.	6	1	3	7·5	Anæmia.	0	2	10·5	7·4	
30	M.	6	2	3	8·4	Anæmia.	1	2	11·4	6·8	
31	M.	35	2	5	6·0	Anæmia.	0	2	12·0	8·5	
32	F.	6	1	2	6·3	Anæmia.	0	1	10·8	7·8	
33	M.	26	3	3	4·2	Lobar pneumonia.	2	3	11·7	8·0	
34	F.	6	2	4	8·9	Anæmia.	1	3	11·7	7·5	
35	F.	4	2	4	6·9	Anæmia.	1	3	10·8	7·8	
36	M.	13	4	5	6·0	Anæmia.	2	3	12·0	8·5	
37	M.	45	5	4	3·9	Anæmia.	2	2	12·7	7·4	Ill several years.
38	M.	30	1	3	4·5	Anæmia.	0	2	11·0	7·5	
39	F.	60	5	3	6·0	Anæmia.	4	1	10·2	8·6	Ill many years.

[1] Fingers' breadth below costal margin.
[2] Hackett's classification.
[3] Grammes per 100 ml.
[4] Grammes per 100 ml.

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Reports of Cases.

POST-DIPHTHERITIC POLYNEURITIS WITH RESPIRATORY PARALYSIS.

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TODAY, diphtheria and its complications are rare, and it is thought that a description of this case and its management would be of interest.

Clinical Record.

A Chinese boy, aged two and a half years, was admitted to the Children's Unit with the following history. Eight weeks previously he had suffered from a sore throat and a fever. He was given two injections by a doctor, but there was no evidence that throat or nasal swabs were taken. Two weeks later he began to regurgitate food through the nose. About four weeks later still he was examined by another doctor, as he was having difficulty in swallowing, and food was still being regurgitated

through the nose. According to the parents he was running about up to the time of his admission to hospital. There was no history of immunization against diphtheria.

On examination, the child was seen to be small; he was not anæmic; his weight was 8·8 kilograms. There was some purulent nasal discharge, and the throat was a little reddened. The heart and lungs were normal on clinical examination. He could sit unaided, but was disinclined to walk. All tendon reflexes were absent except for weak supinator reflexes in the arms. The voice was weak and nasal, and there was some movement of the palate. Coughing was weak and typical of a recurrent laryngeal nerve palsy. Clinical examination revealed no sensory loss. Observation of the child during eating showed considerable nasal regurgitation, but no apparent difficulty in swallowing.

His general condition deteriorated slowly during the first few days. During this time he was fed with thickened feeds through a gastric tube and there was no regurgitation. Difficulty was experienced in keeping the naso-pharynx clear of secretions, and he was nursed prone with a head-down tilt. His cough and voice became progressively weaker and movements of the arms and legs less.

On the morning of the fifth day diaphragmatic movements were nil, there were no movements of the arms and legs, and intercostal movements were weak; the respirations numbered 18 per minute, the pulse rate was 130 per minute and the blood pressure was 130/80 mm. of mercury. There was no cyanosis, but the child was sweating profusely and he was semi-conscious. It was therefore decided to apply artificial respiration, using intermittent positive pressure from a Radcliffe respirator.

Tracheotomy was performed under local anæsthesia, after intubation. It was noticed that the endotracheal tube could be passed easily and without exciting spasm, and that the vocal cords were fixed in the mid position. At the time of commencing intermittent positive pressure respiration (I.P.P.R.) the blood pressure was 140/80 mm. of mercury and the pulse rate was 140 per minute.

Within fifteen minutes of the commencement of I.P.P.R., the child regained consciousness (a marked change from the previous 24 hours) and his colour improved. There were weak movements of the arm and weak attempts to breathe out of phase with the machine. It was considered advisable to keep the child under fairly heavy sedation. An initial dose of 0·15 gramme of thiopentone was given *per rectum*, and thereafter four grains of chloral (0·26 mg) were given every four hours by gastric tube. Sedation was withheld once a day, in order to assess the activity of the child, and as improvement began, sedation was progressively lessened.

Two hours after the commencement of I.P.P.R., the blood pressure was 90/60 mm. of mercury, later rising to 100-120/45-80 mm. of mercury. The pulse rate remained at 130 per minute.

The details of nursing were as follows. The pulse, blood pressure, temperature, respiratory minute volume and respiratory pressure were recorded every half-hour. The patient was turned every hour, and naso-pharyngeal and tracheal toilet was performed. Light percussion of the chest was performed each time the patient was turned, and the nurse in charge was taught to auscultate the chest frequently in order to detect gross changes in the adventitious sounds. This practice was found to be useful, in that the persistence of rhonchi or other adventitious sounds after turning of the patient and percussion was one of the indications for the nurse to get into touch with the doctor.

Feeding was by a gastric tube, 1400 ml. of milk per day being given, fortified to give a calorie value of approximately 1500. Antibiotic cover was given with penicillin and streptomycin.

The Radcliffe respirator was set to give a minute volume of three litres per minute, a respiratory rate of 25 per minute and a pressure of 10 cm. water. Provided rigorous attention was paid to keeping the chest clear, it was found that there was no difficulty in maintaining the required minute volume. There was no difficulty in keeping the lungs clear, and the secretions from the tracheostome remained mucoid and easily aspirated. The blood pressure remained at 100-120/45-80 mm. of mercury, and the pulse rate at 130 per minute for the whole time that the child was on I.P.P.R.

The blood chemistry was investigated on two occasions only. From previous experience in adults undergoing I.P.P.R., it was thought that the over-all clinical picture was a better guide than reliance on repeated biochemical investigations. At the time of the child's admission to hospital, the cerebro-spinal fluid contained 16 cells (lymphocytes) per cubic millimetre, 60 mg. of protein per 100 ml. and 107 mg. of sugar per 100 ml. An electrocardiogram, after three days' I.P.P.R., was normal. The carbon-dioxide combining power at the start of I.P.P.R. was 26 mEq./l.

I.P.P.R. was maintained for two weeks. Voluntary movements began to return five days after it was begun (about 10 days after the onset of paralysis), and thereafter there was a slow but steady improvement. When I.P.P.R. was discontinued, the child was able to swallow and cough and had weak movements of the arms and legs, but could not sit or roll over. Intercostal and diaphragmatic movements were satisfactory. He was finally discharged from hospital some three months after his admission. At that time he was running about, and on clinical examination had full respiratory function, but all reflexes were still absent. During his stay in hospital he gained 2 kg. in weight.

Comment.

Diphtheria is a common disease in Malaya, and the history of the patient before his admission to hospital makes an antecedent diphtheria infection almost certain.

It was interesting to observe that, as respiratory function returned, the child breathed in phase with the machine, and the minute volume tended to rise—sometimes as high as five litres per minute. Although no ill effects were observed from this, the machine was adjusted to maintain a minute volume of three to four litres per minute.

The relatively high blood pressure and pulse rate could not be explained, and these figures fell to normal as soon as unaided respiration began. Hypertension is considered to be a sensitive sign of hypoventilation and carbon-dioxide accumulation. In this case, in spite of a clear chest and an increase in the minute volume to five litres per minute, there was no change in the blood pressure. There was no sweating at any time—another sensitive sign of hypoventilation.

It is thought that the high blood pressure and pulse rate may have been due to vagal paralysis during the acute phase.

Summary.

A case of severe post-diphtheretic polyneuritis is described. Respiration was severely involved, necessitating the use of I.P.P.R. for a short period.

Reviews.

Bacterial and Mycotic Infections of Man. Edited by René J. Dubos, Ph.D.; third edition; 1958. Philadelphia and Montreal: J. B. Lippincott Company. Sydney: Angus & Robertson Limited. 10" × 6¼", pp. 832, with 116 illustrations. Price: 93s. 6d.

THIS book, now appearing in its third edition since 1948, has been almost entirely rewritten because of the amount of new knowledge to be included. Its purpose remains unchanged; it is written for the medical student and the practitioner of medicine. It comprises 36 chapters or essays, written by 37 contributors singly or in collaboration. The first six of these deal with the history of medical

bacteriology, the evolution and ecology of microbial diseases, the morphology, physiology and genetics of bacteria, pathogenicity and defence mechanisms, serology and immunochemistry, and the allergic state; these are followed by 24 chapters on the various groups of bacteria and on human blood groups. There follow chapters on medical mycology, bacteria indigenous to man, principles of chemotherapy, of sterilization, and of epidemiology, and a final chapter on diagnostic bacteriology with an appendix on materials and methods. The book is most readable; the contributors, each an expert in his or her own field, have contrived to pass on the freshness and interest which each of them feels in his subject. This book is not a laboratory manual, but it will be of great use in the laboratory; it is not a systematic textbook, but will be read with more thoroughness and interest than most systematic textbooks. The National Foundation for Infantile Paralysis has given financial support to its preparation, so that while the type, paper and general format are superlatively good, the price is relatively low. The chapter by Dubos on "The Evolution and the Ecology of Microbial Diseases' is a fascinating one. Dubos has "tried to express the view that the ability of microorganisms to produce pathologic changes is under the influence of large biologic forces as yet poorly understood which do not manifest themselves in the form of recognized immunochemical reactions". There speaks the true philosopher and scientist, whose vision goes past the small temporal suburbs of our present wavering knowledge to the infinite universe beyond. This is a book to be bought, read and kept.

An Introduction to Surgery for Dental Students. By R. P. Jepson, F.R.C.S., and B. N. Catchpole, F.R.C.S.; 1959. London: The English Universities Press, Limited. 8¾″ × 5¼″, pp. 176, with 53 illustrations. Price: 39s. 3d.

EMPHASIS on the fundamental principles underlying surgery is a feature of modern teaching. Now for the first time we find this applied in the teaching of surgery for dental students in this small volume. It is based upon lectures which deal with general principles rather than the details of the special techniques of oral surgery. It can be recommended confidently to dental students and practising dentists.

First of all, there is nothing in the book which dental students should ignore. All highly academic discussions have been omitted. The book is essentially practical. One can think of relatively unimportant subjects which could have been included; but it was probably wiser to keep the volume small and thereby more attractive. Various aspects of the physiology and pathology of wounds, of course, are of concern and interest in any form of surgery. One finds also a clear and simple description of all those aspects of regional surgery which can, in any case, impinge upon the work of the dental surgeon.

The subjects discussed must, of course, have been influenced by the scope of the students' lectures in oral surgery; but we think it unlikely that those would have included respiratory obstruction, and this could well have been included in this book. Nevertheless, as a small textbook it is highly recommended.

Fracture Surgery: A Textbook of Common Fractures. By Henry Milch, M.D., and Robert Austin Milch, M.D., with a chapter on anæsthesia by Herbert D. Dubovsky, M.D.; 1959. New York: A Hoeber-Harper Book. 10″ × 6¾″, pp. 480, with 480 illustrations. Price: $17.50.

THIS new book of 470 pages has been designed to attempt to bridge the gap between the small handbook of fracture management and the encyclopædic volume for the specialist. From the preface to the book, it is especially aimed at helping the resident medical officers in dealing with problems in the casualty ward.

It is arranged in two sections, the first dealing with general considerations of fracture management, and the second with specific fracture management affecting individual bones.

It is copiously illustrated, but rather poorly so. The X-ray reproductions are in many cases indifferently reproduced, and frequently are not particularly helpful in illustrating the accompanying text. The line drawings, too, cannot be compared with the beautiful examples we have become used to in good modern medical literature.

It is a curiously uneven book, and one cannot help wondering whether the authorship varies from chapter to chapter between the elder and the young Milch.

In the first section there is a very useful chapter on the authors' concept of axial malalignment of fractures. This chapter should be of real value to the young surgeon, and contains some interesting new ideas, particularly on X-ray interpretation of fractures. Another chapter on plaster of Paris is far too detailed and unnecessarily academic. Otherwise this section is mediocre.

The second section is more useful to the young surgeon; but one cannot agree with all the views expounded or techniques described, and the authors are rather dogmatic in their views, to the exclusion of other generally accepted methods in several instances.

The chapters dealing with fractures of the upper extremities are generally very good and helpful. However, one finds many divergent views, from other methods of dealing with fractures of the lower extremity. This is particularly so in regard to the hip region. In this section, too, one must criticize the authors, who describe their so-called resection-angulation operation for ununited femoral neck fractures, with no words of acknowledgement to Girdlestone or Batchelor.

This book cannot be recommended as a textbook on fractures for the resident medical officer. There are some excellent chapters and some very helpful new conceptions. However, this level is not maintained uniformly, and the information must be sifted with some experience and discrimination, which detracts from its value as a book to be kept in the casualty department library.

Orthopædic Surgery. By Sir Walter Mercer, M.B., Ch.B. (Edin.), M.Ch.Orth. (Liv.) (Hon.), F.R.C.S. (Edin.), F.A.C.S. (Hon.), F.R.C.S. (Eng.) (Hon.), F.C.S.S.Af. (Hon.), F.R.C.S.E. (Hon.), F.R.S. (Edin.); fifth edition; 1959. London: Edward Arnold (Publishers) Limited. 9″ × 5¾″, pp. 1076, with 422 illustrations. Price: 90s. (English).

THE worth of this very well-known textbook can be gauged from the fact that, first printed in 1932, it has now reached the fifth edition, as well as having had four reprints during this period.

As a result of further amplifications, the book now comprises 1075 pages. Although most of the old material has been retained with little alteration, the chapters on congenital dislocation of the hip and other congenital affections, scoliosis, the paralyses, malignant disease and other tumours of bone and general affections of the skeleton have been rewritten entirely, and many other chapters extensively revised. The new material is particularly well set out, notably that dealing with congenital dislocation of the hip and scoliosis.

This has proved such a dependable and worthwhile orthopædic textbook over the years, particularly for the student and young graduate, that it is difficult to criticize it to any extent. The illustrations on the whole are good—though some are becoming dated—and plentiful. The legend is in rather small type; but we appreciate that it would be necessary to divide the book into two volumes if this was not so, which would, of course, increase the cost to the already overburdened student.

If we must find fault with the individual items, which is difficult, we might suggest that the detailed tabulation of the differential diagnosis of sacro-iliac strain and lumbo-sacral strain is more academic than real. A similar criticism can be levelled at other rather simplified tabulations of conditions which the practising orthopædic surgeon would find much less clear cut and well defined—for example, in dealing with postural deformities, scoliosis, etc. However, we realize that this, if it is a fault, is probably necessary in presenting a clear picture to the student. For this same reason the methods of treatment are often rather glibly dealt with, with no hint of probable deficiencies in our knowledge or the results of treatment.

These are but minor criticisms of a preeminently satisfactory and complete textbook of orthopædic surgery for the undergraduate and recent graduate, and a book which most experienced surgeons have found most useful to have on their shelves.

International Textbook of Allergy. Edited by J. M. Jamar, M.D.; first edition; 1959. Oxford: Blackwell Scientific Publications. 10¾″ × 7″, pp. 639, with illustrations. Price: £5 10s. (English).

THIS textbook brings the subject of allergy to us in the modern manner. The diversities of the subject have grown so much that experts with long training and experience in particular facets of allergy have been chosen to contribute a chapter. The international flavour is given by contributors from eight European countries and both Americas. Because of this, there is some repetition of fact, and also some difference, both of experience and of opinion.

This book should be on every physician's shelf and in every hospital library for it contains, in detail, all the present theories, hypotheses and facts relating to the basic principles of the subject. This information is well set out and, mostly, simply explained. Clinical application of theory is plentiful.

The first half of the book is the better, particularly in the chapters on general subjects—for example, "Allergy and Immunology", "Fungi and Miscellaneous Inhalant Allergens", "Reactions to Drugs"; whilst those chapters relating to a specific segment of allergy, particularly those on "Status Asthmaticus" and "Allergic Diseases of the Eye" do not read so well. However, there is an abundance of common sense and a colossal amount of useful factual detail throughout.

The book is not entirely suitable for student or general practitioner reading, but as a work of reference it would be most useful. If any practitioner has a yearning for information on the management of difficult cases, he would find the answers to most of his queries here. There are answers to everything; but still, many of the authors reveal that there are patients who remain problems in spite of the application of the best principles of therapy.

Perhaps one quotation will give an estimate of the advanced thinking and originality of this book:

> Studies which we have carried out since 1951 on more than 240 patients suffering from "intrinsic bronchial asthma' have led us to believe that the characteristic triad—nasal polyps, bronchial asthma, and aspirin sensitivity—which develops, late in life, in non-allergic individuals, might be based on a cholinergic, rather than a histamine-releasing mechanism. It is our impression that this group of patients represents a separate entity which, although clinically similar to bronchial asthma of allergic origin, is a disease of different etiology and of a different course. If our reasoning is correct, this particular syndrome which originates at the receptor sites of the indicator tissues would be the only truly "intrinsic" bronchial asthma.

This book is a definite advance on any of its predecessors on this subject. We hope that it will be reissued from time to time, since the contributors would have little difficulty in reediting and modernizing their own particular chapters, and then the "International Textbook" would remain always up to date.

British Obstetric and Gynæcological Practice. Edited by Sir Eardley Holland, M.D. (Lond.), F.R.C.P., F.R.C.S., F.R.C.O.G., and Aleck Bourne, M.A., M.B., B.Ch. (Cantab.), F.R.C.S., F.R.C.O.G.; "Obstetrics" edited by Sir Eardley Holland; second edition; 1959. London: William Heinemann, Medical Books, Limited. 9¼" × 7", pp. 1242, with many illustrations and 24 tables. Price: 147s. (English).

THE first edition of this book appeared in 1955, and, as its editors stated, their guiding principle was conservatism. This principle is evident throughout an enlarged and extensively revised second edition. Almost all the 42 contributors are obstetricians practising in the British Isles; so the volume is essentially a practical exposition of conservative obstetrics and as such is highly recommended.

New chapters have been added on fœtal physiology, and an up to date account is given of recent work on the variations in the functional efficiency of the placenta due to aging or to maternal disease. An excellent chapter has been added, discussing the changes undergone by the infant during its adaptation from easeful intrauterine to a hazardous extrauterine life. No less than 125 pages are devoted to diseases associated with pregnancy, and this is a most valuable section of the book. Heart disease in pregnancy and the anæmias in pregnancy are described particularly well.

In general, the principles laid down and the practical methods of treatment advocated in this volume are remarkably paralleled by the best practice in this country. So much is good that one hesitates to make minor criticisms; but a few may be stated. In outlining the procedures of ante-natal care, greater emphasis could be laid on the significance of blood pressure readings. In this section, this most important feature of ante-natal care earns a mention only on the ante-natal record form.

In the section on toxæmia of pregnancy, it is stated that hypertension is the earliest unequivocal sign of preeclampsia, and that if the systolic blood pressure exceeds 140 and the diastolic 90 mm. of mercury, and provided that neither is below these figures, hypertension exists. This criterion would not be acceptable to most Australian obstetricians, who have come to realize that the blood pressure level is a characteristic of the individual, and that in the diagnosis of preeclampsia the all-important thing is a persistent increase above that individual level, whatever that figure may be.

In the discussion on essential hypertension, the vexed question of what constitutes the upper level of blood pressure "normalcy" is not resolved. Although we would agree with the statement that "if the blood pressure is elevated to 140/90 on two or more occasions before the twentieth week of pregnancy" essential hypertension is diagnosed, that level is higher than is taught in many centres.

In a series of photographs, the delivery of an infant with the breech presenting is well shown, from the time the breech "peeps" till the after-coming head is delivered. In the description of the methods of delivering the after-coming head, the use of forceps is mentioned only as a method of dealing with an abnormal arrest of the head. In many clinics, this method of delivery has come to be much more generally used.

It is interesting to read that the operations of lymphysiotomy and pubiotomy are enjoying one of their periodic waves of revival. As the author states, very few obstetricians in Britain or the United States advocate these operations, for it is indeed difficult to see their advantages over Cæsarean section.

There is a useful chapter on medico-legal aspects of obstetrics.

The printing, illustrations and general set-up of the book are a credit to the editor and the publisher. It is a volume that should be in easy reach of all who practise obstetrics, for within its covers will be found a sound answer to many problems of management that worry all obstetricians.

Principles of Radiation Dosimetry. By G. N. Whyte; 1959. New York: John Wiley & Sons Inc. London: Chapman & Hall Ltd. 9" × 5¾", pp. 131, with illustrations. Price not stated.

THIS book deals with ionizing radiations only, and has been written for the reader with a reasonably sound background of atomic physics and mathematics. There is no reference to ultra-violet and other non-ionizing radiations. On the whole the material is well presented and the references are very extensive and well chosen, adding considerably to the value of the book. The main criticism is that it is too short for a subject which requires detailed discussion, and in too many instances the author has left the reader to look up the references for a full appreciation of the point being made.

The approach to the subject is predominantly theoretical, and the practical problems involved in dosimetry could well have been discussed more fully. Chapter 3, on the determination of X-ray spectral distributions, is only a very general account occupying less than five pages, and the very important subject of dosimetry of charged particles deserves much more than the few pages devoted to it. For those concerned with the biological and medical applications of radioisotopes and health physics, one serious omission is a discussion on the complex but very important dosimetry problems of internally administered radioisotopes.

In spite of these drawbacks, however, the book can be recommended as a good starting point for understanding the nature and difficulties of dosimetry problems. The discussion on the interaction of radiation and matter, which is very fundamental to the subject, is dealt with in a comprehensive manner and is the outstanding section of the book.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Variations on a Theme by Sydenham: Smallpox", by P. B. Wilkinson, M.R.C.P.; 1959. Bristol: John Wright & Sons Limited. 9¾" × 7¼", pp. 76, with 69 illustrations. Price: 17s. 6d. (English).

"Medical Helminthology", by J. M. Watson, D.Sc. (London), A.R.C.S.; 1960. London: Baillière, Tindall and Cox. 9¾" × 7¼", pp. 490, with illustrations. Price: 84s. (English).

"Encyclopedia of Medical Syndromes", by Robert H. Durham, M.D., F.A.C.P.; 1960. New York: Paul B. Hoeber Inc. 9¼" × 6", pp. 644. Price: $13.50.

The Medical Journal of Australia

SATURDAY, MAY 7, 1960.

DRINKING DRIVERS.

EVERYONE with any sense of responsibility deplores road accidents resulting from the excessive intake of alcohol. What is too often overlooked is the vital importance of preventing them. It is all very well to punish those responsible when the damage has been done, but these accidents should never happen and seldom would if sufficiently bold measures were taken. The fundamental fact that is still too little acknowledged in practice, whatever lip-service may be given to it, is that drinking and driving do not mix. This is a straightforward physiological fact. The effect of small amounts of alcohol may not be significant for many drivers, and some can tolerate more than others, but there is a level at which alcohol makes anyone unfit to drive a motor vehicle. The sensible thing then is to try to see that he does not do so. We can educate him, frighten him, forbid him or block him, the approaches are many, but in one way or another the drinking driver must be kept away from his vehicle. Anyone who doubts the importance of this is invited to study the article by J. H. Birrell published in this issue (see page 713). Based on extensive practical experience in the Victorian Police Department and the careful sifting of a mass of material, it warrants the fullest respect. It is impossible to ignore it.

At the same time attention is drawn to the report recently issued by the Victorian Government Pathologist, K. H. Bowden, on the basis of his investigation of the problem overseas. This report was reviewed in some detail by the Melbourne *Age* on April 8, 1960. Its keynote is prevention, and a number of useful ideas, new to this country, are brought forward. In New Zealand, for example, the police have the power to take the keys away from a person whom they think unfit to drive a vehicle, if it appears that he is about to do so. The public, Dr. Bowden states, have accepted this legislation, and relations between police and public have been good. In Belgium a law forbids a person to drive a car within two hours of a policeman's warning that alcohol has rendered him unfit. In Copenhagen also the police have the power to forbid an unfit motorist to drive. In Canada two lesser charges have been introduced—having control of, and driving, a vehicle with ability impaired by alcohol. These charges carry less severe punishment, but are easier to substantiate than the more serious type of charge, and their deterrent effect is regarded as being so much the greater. Another approach to the problem is the provision of beer with a lower alcoholic content than that at present available in Australia, an idea which has been canvassed by thoughtful people here for a good long time, with conspicuous and puzzling lack of success. In Norway they apparently have no difficulty in providing three grades of beer; it should be possible in Australia. The strength of the police force is of course of great importance in any programme to prevent accidents due to excess of alcohol, as with those from many other causes. This fact is well recognized in, for example, Sweden and Denmark, and it needs more consideration in Australia; as Dr. Bowden says, no matter what the legislation, in a democratic country such as ours, the number of arrests for drunk driving must depend directly on the activity of the police, and not on fellow citizen reporting fellow citizen.

On the question of medical examinations, Dr. Bowden recommends that, if the voluntary system is retained in Melbourne, an independent panel of doctors for examination would be more in keeping with overseas practice. He refers to a strong feeling in Britain that medical examination should be retained for the protection of the suspect, who may be suffering from some other condition, with symptoms simulating alcoholism. On the other hand, medical practitioners are not needed to operate a "breath-alyzer" programme, which can be handled by technicians. Breath tests are apparently widely used in Canada and the United States and were commented on favourably by the B.M.A. Special Committee whose report we reviewed a short time ago.[1] They are not looked on with favour in Europe because of their inaccuracy, but Dr. Bowden considers that, used intelligently, a breath-testing programme has much to commend it when it can be suitably adapted to legislation in force. If breath tests are adopted and given their proper place in the chain of evidence, there seems no good reason why they should not be compulsory.

A good deal of attention has been given to the question of blood alcohol estimations. In most States of the U.S.A., 0·15% of alcohol in the blood is prima-facie evidence of driving in an intoxicated state. In Norway and Sweden a level of 0·05% constitutes an offence. Compulsory physical examination and blood testing are provided for by legislation in Norway, Sweden, Denmark, Germany, France, Belgium and Switzerland. Dr. Bowden points out that this compulsion has a number of benefits: evidence against suspects is more complete, more offenders are punished, laws are more easily enforced, and (a fact often overlooked) an innocent person is unlikely to be wrongfully convicted. The weakness of voluntary chemical-test legislation has become more obvious with experience. In New York, where chemical tests are voluntary, the conviction rate when chemical tests have been used is about 84%, whereas from those who refuse a test it is 66%; in the light of this it is not surprising that there is an increasing reluctance to submit to the tests. Dr. Bowden

[1] MED. J. AUST., 1960, 1: 299 (February 20).

recommends that if it is intended to retain the legislation for voluntary tests in Victoria, consideration should be given to lowering the level constituting prima-facie evidence of being under the influence of intoxicating liquor, and there might be a reexamination of the question of the defendant's refusal to submit to a test being admissible evidence.

Opinions are still divided in Australia on the advisability of having compulsory blood tests. It is of course a legislative rather than a medical matter, and the Federal Council and some of the Branch Councils of the B.M.A. have felt that it is not in their province to express an opinion on it. This is a reasonable view, though debatable. However, one wonders if the objections sometimes raised to compulsory tests are valid in the face of the major problem of public health and community welfare that emerges so starkly from Dr. Birrell's study. Examination of public health legislation in any of our States or in the Federal sphere will reveal plenty of instances of intereference with individual liberty in the common interest just as great as that involved in compulsory blood testing for alcohol. Moreover, if the results of blood tests are kept in perspective and not accepted as absolute evidence, objections based on their variability and difficulty of interpretation largely disappear. Certainly the innocent driver has nothing to fear from them.

A SIGNIFICANT APPOINTMENT.

Major-General W. D. Refshauge, at present Director-General of Army Medical Services, is to succeed Dr. A. J. Metcalfe as Commonwealth Director-General of Health in June. This appointment, announced recently by the Commonwealth Minister for Health, Dr. D. A. Cameron, is of considerable interest and will be generally approved. General Refshauge, who graduated in medicine from the University of Melbourne in 1938, served with distinction in the Army Medical Services throughout the second World War. In 1946 he joined the staff of the Royal Women's Hospital, Melbourne, and was medical superintendent of the hospital from 1948 to 1951. He became a Member of the Royal College of Obstetricians and Gynæcologists in 1947. Having returned to the Army Medical Services in 1951 as Deputy Director-General, he succeeded Major-General Sir Kingsley Norris as Director-General in 1955.

General Refshauge's transfer from his Army post to take charge of the Department of Health is evidence of a vigorous Government policy in the general sphere of health and medical administration. It is apparent that the Government desires to have greater coordination and a more intimate link between the Department of Health and other medical services—especially those of the Defence Forces. It is understood that General Refshauge is to be available to the Department of Defence, as required, to assist in the furtherance of the present policy of greater coordination and integration of the Armed Forces Medical Services. Some worthwhile gains along this road have already been made in the Department of Defence through its Medical Services Committee, which comprises the three medical directors-general of the Armed Forces and Professor S. Sunderland. The present appointment will do much to further these important and constructive changes. Future developments will be watched with great interest by the medical profession. One may also hope that the appointment will help to strengthen and augment existing links of mutual understanding and trust between the practising medical profession and the Department.

Current Comment.

RESTLESS LEGS.

> . . . a common minor ailment, which I have never seen described. It occurs most often when the legs get warm in bed, and prevents going to sleep. It has no relation to the sudden leg-jerk which sometimes occurs at the moment of going to sleep. A curious unlocalized restlessness is felt in one or both legs. It is not quite a pain, but is distinctly unpleasant. There is no skin irritation. . . . Massage has no effect, but getting up and walking about may give relief. In ten minutes to an hour the "jitters" depart, and sleep comes.

This description by F. G. Allison[1] of Winnipeg refers to a condition which must be familiar to many as a personal experience, but which is rarely mentioned in the consulting room. Textbooks, with few if any exceptions, make no reference to it. It is one of those obscure conditions which have been neglected, not because they are rare, but because the symptoms experienced are strictly subjective, there are no clinical signs, and the patient's general health is, as a rule, unaffected. (Another example of such a condition is proctalgia fugax, one which is probably not nearly so common as restless legs but which has received more attention because of its more dramatic nature.) The earliest identifiable reference to the syndrome of restless legs is stated by E. Ask-Upmark[2] to have been by Thomas Willis, an English physician who flourished in the mid-seventeenth century. During the next 300 years it is probable that passing references to the condition were made by various medical writers, but these are not easy to trace. In a leading article in *The Journal of the American Medical Association*[3] it is stated that Wittmaak gave a description of a similar syndrome in 1861, in which he "referred to a condition described by older physicians as 'anxietas tibiarum'", a name so apt that it is difficult to doubt that it was applied to the same condition. In 1936, R. Scot Skirving[4], in a paper "On Herpes, Cramps, Fidgets and Other Disordered Sensations", was undoubtedly thinking of the same condition in his remarks on fidgets. However, apart from Allison's brief account, little was heard of the syndrome until 1945, when A. Ekbom drew attention to it in a paper in which he described a long series of cases in patients seen in Stockholm between 1943 and 1945. Since then several further papers on the subject have been published by Ekbom and other Swedish physicians, though the condition appears to have received little notice in the English-speaking world. According to Ekbom about 5% of the adult population experiences these symptoms, and their incidence is over 10% among pregnant women. These paræsthesiæ are apparently experienced in various forms by different people; to some they are definitely painful, and in his original description Ekbom distinguishes four types of the condition, ranging from cases in which the sensation is purely paræsthetic to those in which it is purely painful, though the latter appear to be rare. In the great majority of patients these symptoms cause no real disability. Allison, himself a sufferer, states: "I have observed this condition mainly in myself, relatives, and friends, as patients do not complain of it unless it occurs frequently enough to cause loss of sleep." However,

[1] *Canad. med. Ass. J.*, 1943, 48: 38 (January).
[2] *Acta med. scand.*, 1959, 164: 231 (fascicule 3).
[3] *J. Amer. med. Ass.*, 1945, 129: 875 (November 24).
[4] *Med. J. Aust.*, 1936, 1: 645 (May 9).

in a few patients the condition is severe, and Ekbom has collected a number of these, including one extreme case in which the patient had undergone unilateral lumbar sympathectomy without obtaining relief. The characteristic feature of the paræsthetic form of the condition is that it comes on only when the patient is at rest and is always relieved by movement. (The fact that the painful form is said also to occur when the patient is walking about makes one wonder whether this is in fact another condition.) Ekbom has pointed out that the paræsthesiæ may occur when the patient is sitting in a theatre, and in fact some patients experience them mainly under such conditions. Some writers have attempted to equate restless legs with other pathological conditions, such as meralgia paræsthetica or myokymia, but there is little difficulty in showing that these authors have misunderstood the nature of the condition. W. F. T. Tatlow[5] of Montreal, one of the few English-speaking writers to have reviewed the condition, gives a clear account of it, in which he points out the distinctions from acroparæsthesia and burning feet. The pathogenesis of the condition, which may last the greater part of a lifetime, is obscure. Allison obtained prompt relief by chewing a tablet of nitroglycerine. Ekbom treated his patients with vasodilators such as "Priscol" or "Carbachol", with varying success. N. B. Nordlander[6], noting that some of his patients were anæmic, treated them with intravenously administered iron-dextran compounds. This was extremely successful, whether or not the patients were anæmic. He then tried injection of 20 ml. of 10% dextran solution alone, with prompt success in the first patient, but without result in a second. A third patient, who had not responded to all other forms of therapy, obtained prompt and lasting relief after an intravenous injection of heparin. Nordlander attempts to correlate these results by suggesting that these remedies work by blocking the reticulo-endothelial system. The latest contribution on the subject is a short paper by Ask-Upmark[3] in which he points out that partial or complete relief may be obtained by lying in a prone position. He makes the very plausible suggestion that the cause of the condition is a postural one, and that it is the result of engorgement of the venous system in parts of the spinal canal. It is not unlikely that the symptoms can be produced by several different causes; for example, they have been described in connexion with avitaminosis in prison camps. However there appears to be a very large body of idiopathic cases for which no simple explanation is available. The condition is probably a true entity. In spite of the somewhat chequered therapeutic story, it is probably not primarily of neurotic origin, in view of the very large number of otherwise normal people who experience these sensations, though it is likely that the symptoms may be readily exaggerated in a neurotic subject. Finally, in view of the elusive nature of the symptoms, it is probably safe to say that most of those who have described the condition accurately are themselves sufferers from it.

PROBLEMS OF AGING.

THE number of old people is increasing in most countries, partly as a result of successful campaigns against disease. Some of them suffer from mental illness, even more are lonely or poor, and a good many support all these disabilities at the same time. In view of the growing urgency of these problems, the attention of the sixth WHO Expert Committee on Mental Health which met in Geneva last year, was devoted to the aging and the aged.[1]

The report shows that suicide statistics for the aged are high in both the industrialized countries and those that are in the course of development, an indication of the universality of the problem. The largest number of suicides are in men of 70 years and over in Belgium, France, Italy, Holland, Portugal, England, Australia, Switzerland and Spain. In Canada, Norway and Sweden the highest rates are in the 60 to 69 years age group and in Denmark in that from 65 to 74 years. Where women are concerned, most suicides occur approximately ten years earlier than for men. Suicides are not rare in a good number of the economically less developed countries. Where inquiries have been made—for example, in Hong Kong—they indicate that the chief causes are unemployment, disorganization following political changes, poverty, physical and mental illness—as in the more highly developed countries. Observations confirm that in the under-developed countries old people are particularly affected by rapid social change. Very often old people who commit suicide have also suffered from mental and physical difficulties that could have been cured or alleviated. In most countries the suicide hazard is greater for single, widowed or divorced persons than for those who are married, and there is a tendency for the aged to commit suicide in isolation.

The many explanations for this state of affairs should be considered as part of the total economic and social picture. But first it must be realized that if the aging of a population creates new problems, it also reflects progress. Most people at the present time can look forward to a longer and more rewarding life, and many old people, although without remunerative employment, continue to be useful to society. Moreover, in the countries that are most highly developed from the public health point of view, the proportion of invalids among the aged has undergone a progressive reduction in the last thirty or forty years. The reasons for the greater frequency of mental illness must be sought in present-day society itself, but without our postulating that this is greater than it used to be. For today the psychiatric hospital frightens no one; admissions are higher, a good sign, because most people who are treated are assured of being cured.

Although the loosening of family ties has been blamed for the present situation, studies have proved, to the contrary, that affection of children for their parents has survived in almost all societies. It is, nevertheless, true that industrialization and urbanization encourage social and geographic movement, with the result that a certain number of old people find themselves deprived of human contact, especially in big cities. Between 10% and 20% of the aged are said to be isolated in this way. As with suicides, bachelors and people living alone represent the largest number of persons admitted to psychiatric hospitals. Providing more human contact for those who have no relatives and friends ought to be one of the primary objectives of all preventive measures taken by the community. It is also noted that in certain countries, more of the economically underprivileged are admitted to psychiatric hospitals than those who are well off, and more city people than those from the country.

Taking these factors into account, the WHO Expert Committee decided to define the methods by which this situation could be improved. In addition to purely health measures, such as the organization of mental health services especially adapted to the problems of old age, in clinics and at home, the Committee recommended a certain number of socio-economic measures designed to lessen the instability of old people by employment and the institution of old-age pensions where they still do not exist, by legal assistance to help them settle their affairs, by social activities for the aged and by maintaining old people in their homes if possible—that is to say, in the places where they have lived all their lives. The Committee also recognized that it was not enough to adopt such measures, no matter how efficacious; it was still necessary to teach old people the art of growing old by every means possible and to modify the attitudes of society towards them. The wisdom and experience of old people represent a precious heritage for younger generations. Well-balanced and well-adapted old people are a very useful element in human society. In their interest and that of their children they should be assured of a worthy life.

[5] *Canad. med. Ass. J.*, 1954, 71: 491 (November).

[6] *Acta med. scand.*, 1953, 145: 453 (fascicule 6).

[1] "Mental Health Problems of the Aging and Aged", WHO Technical Report Series, No. 171; 1959. World Health Organization: Geneva.

Abstracts from Medical Literature.

PHYSIOLOGY.

Requirements of Sodium Chloride during Summer in the Tropics.

M. S. MALHOTRA, B. K. SHARMA AND R. SIVARAMAN (*J. appl. Physiol.*, September, 1959) present data on salt requirements in the tropics, based on observations on 24 acclimatized Indian subjects given diets containing 16·2, 11·2, 8·7, 6·2 and 3·1 grammes of salt per day. Adequacy of salt diet was tested from the chloride excretion in urine and from the changes observed in thiocyanate space and plasma chloride concentration at the start and after the subjects had been on the restricted salt diet for a week. Sweat and chloride losses at different environmental temperatures were studied. The salt requirement of subjects walking for two hours in the sun at a speed of about 3·5 miles per hour was found to be about 6·2 grammes per day when the mean maximum environmental temperature was 100·7° F. The requirement was found to increase by 0·063 gramme for each degree rise in temperature (Fahrenheit).

Trophic Maintenance of the Diaphragm.

N. C. JEFFERSON *et alii* (*Amer. J. Physiol.*, September, 1959) report that in dogs the central end of the cut phrenic or vagus nerve was anastomosed to the distal end of the phrenic nerve. After phreno-phrenic anastomosis, slight atrophy of the muscle and nerve elements of the diaphragm occurred up to seven or eight weeks, followed by regeneration. At that time, electric stimulation of the nerve above the anastomosis produced contraction of the diaphragm, although respiratory function was absent. Ten to 11 weeks after anastomosis, spontaneous respiration recurred. In the case of vago-phrenic anastomosis, spontaneous respiration did not reappear within 58 weeks, but stimulation of the vagus above the anastomosis produced contraction of the diaphragm, and histologic study showed regeneration of muscle and nerve elements. It is concluded that trophic maintenance of the diaphragm depends on the presence of functionable innervation, not necessarily on functioning innervation.

The Ciliary Activity of Human Respiratory Epithelium.

G. CORSSEN AND C. R. ALLEN (*J. appl. Physiol.*, November, 1959) report that explants of human respiratory ciliated epithelium cultured in a plasma clot tend to round up and to form rotating globes which can be employed in perfusion chambers for the study of the effect of various chemicals on ciliary activity. Perfusion of acetylcholine chloride solutions at concentrations of 0·1% to 1·0% invariably increased rotatory movement of the cell cluster. Bathing the epithelial explant in solutions of 0·05% to 0·1% eserine salicylate, prior to the perfusion with acetylcholine chloride, markedly enhanced the effect of acetylcholine on ciliary activity. Perfusion of atropine sulphate solutions with a concentration of 0·1% or stronger resulted in a short-lasting stimulatory effect, followed by progressive reduction of rotatory motion. The depressant action of atropine sulphate was counteracted by acetylcholine chloride at 0·5% concentration. The behaviour of the various substances investigated appears to be consistent with the assumption that acetylcholine plays the key role in initiating and maintaining ciliary motion.

Thyroid State and Working for Heat in the Cold.

V. G. LATIES AND B. WEISS (*Amer. J. Physiol.*, November, 1959) report that four experiments were conducted in which rats in a cold room were allowed to obtain a burst of heat from a heat lamp by pressing a lever. When working at 2° C., hypothyroid rats began to work for heat at a steady rate earlier in a sixteen-hour session than did euthyroid rats. This rate itself was both higher and more steady for hypothyroid than for euthyroid rats. Euthyroid and hypothyroid rats working at 5° C. showed a difference only in time required to attain their steady rate. In one experiment, the chronic administration of l-triiodothyronine to hypothyroid rats led to a significant decrease of lever presses as compared to the performance of normal rats. Discontinuance of l-triiodothyronine led to a gradual recovery of the high rate. The differences in this kind of behaviour between hypothyroid and euthyroid animals were attributed to differences in drive state arising from the tendency of body temperature of hypothyroid animals to decline more rapidly in the cold.

Deposition and Fate of Cholesterol.

M. FRIEDMAN AND S. O. BYERS (*Amer. J. Physiol.*, November, 1959) report that aortic implants were placed in the anterior eye chamber of 30 rabbits subsequently fed excess cholesterol and cottonseed oil. After three months of such feeding, seven of these animals were sacrificed and their implants and also segments of their own aorta were analysed for cholesterol. Two more groups of these rabbits were sacrificed two and three months respectively after their return to a cholesterol-free diet. It was observed that the aortic implant gained almost twice as much cholesterol as the host's own aorta at the end of the cholesterol feeding period. This difference became even greater in animals which were sacrificed two months after cessation of excess cholesterol feeding, but were still hypercholesterolæmic. However, the ocular implants of rabbits examined three months after cessation of cholesterol feeding were observed to have lost almost all of their cholesterol, whereas the animal's own aorta continued to exhibit an unchanged excess of cholesterol. The findings suggest that the ocular aortic implant differs markedly from the aorta *in situ* in regard to its penetration and retention of cholesterol.

Positive Pulse Reflection.

W. F. HAMILTON AND W. J. BROWN, JR. (*Amer. J. Physiol.*, October, 1959) state that earlier work from their laboratory has demonstrated that a standing or resonant wave is set up in the dog's aorta which causes simultaneous augmentation of the systolic pressure in the lower parts of the aorta. It has been suggested that this is due to reflection of the pulse wave from the peripheral arterioles. The possibility of such reflection has been denied by various authors on the grounds that the aggregate cross-sectional area of the arterioles is greater than that of the aorta, and that reflection in such a region should be negative rather than positive. A model has been constructed of a distensible rubber tube, an end of which can be open or closed with a stopper or a bundle of capillary tubes with aggregate cross-sectional area larger and resistance to flow greater than those of the distensible tube. With tube distended, pulse-wave trains were generated in the tube with the end either open or closed. Pulse waves at the two ends of the tube were recorded simultaneously, and recordings showed that with the open tube negative reflection took place, whereas with the tube closed the reflection was unequivocally positive.

Estimation of Body Surface Area and Volume.

L. SENDROY, JR., AND L. P. CECCHINI (*J. appl. Physiol.*, November, 1959) describe a convenient and rapid photographic technique of obtaining data which can be used for the calculation of human body surface area. The results, which are in good agreement with values obtained by a reliable method of readings from a chart, provide additional support for the application of the increasingly important photographic method of quantitation in human biology. Data have also been obtained which suggest that the surface area of dogs may satisfactorily be estimated by the same previously reported chart method used for human beings. Empirical equations for the calculation of body volume (and density) in man, based essentially on measurements of weight and height, have been developed and tested in respect to measured values obtainable from the literature. Statistical evaluation and the criteria of convenience and rapidity in use, rather than more restrictive theoretical considerations, indicate the superiority of predominantly empirical relationships as the methods of choice for the prediction of body volume. Comparison of the reliability of the results with those obtainable by established methods of quantitation indicates that these equations may be useful as approximate, but most convenient, indices of gross body composition.

Nerve Conduction Block Produced by Oxygen.

P. L. PEROT, JR., AND S. N. STEIN (*Amer. J. Physiol.*, December, 1959) report that oxygen at high pressure causes a block of conduction in the peripheral nerves of mammals. The interval between initiation of exposure to high oxygen pressure and the onset of block is inversely related to the partial pressure of the oxygen. If the block is not maintained for more than a few minutes, recovery of the ability to conduct returns to the nerve upon decompression. The sensitivity of mammalian nerve to the toxic effects of oxygen at high pressure is decreased when the partial pressure of carbon dioxide in the atmosphere is

high, whereas if the partial pressure of carbon dioxide in the atmosphere is lower than normal, the sensitivity of the nerve to the effects of high pressure oxygen increases. This is contrary to the effect carbon dioxide has on oxygen toxicity in frog peripheral nerves. Increased metabolic activity, as occurs at high stimulation frequencies, shortens the time required for block; decreased metabolic activity, as occurs when nerves are cooled, delays the onset of block.

Evidence for Left Ventricular Diastolic Suction.

H. SCHEU AND W. F. HAMILTON (*Amer. J. Physiol.*, December, 1959) report that the question of ventricular filling by "suction" was investigated by recording the transmural ventricular pressure in normally breathing animals which had recovered from surgery. This transmural pressure was recorded as the difference between the thoracic pressure and the intraventricular pressure. It was considered positive whenever the ventricular pressure exceeded the thoracic pressure as is the case during systole, and negative when it was less than the thoracic pressure. During diastole the difference between these pressures is always small (±10 mm. of mercury or less. In the normal untraumatized animal it is zero or positive (filling by venous pressure). After the animal deteriorates from repeated experiments, it may become negative (filling by an action of the ventricular wall or suction). This suction is greater as a result of conditions which interfere with venous return and diminish the size of the ventricles, as determined by X-ray examination (e.g., mitral stenosis or bleeding). The force of suction is lessened after transfusions which increase the size of the ventricles. Considerations are advanced for believing that the suction force originates in elastic recoil of the ventricular wall and that it is not clearly an important factor in the mechanism of cardiac pumping.

BIOCHEMISTRY.

Ethanol Metabolism.

M. E. SMITH AND H. W. NEWMAN (*J. biol. Chem.*, June, 1959) have studied the rate of ethanol metabolism in fed and fasting animals. Liver slices from fasting rats metabolize alcohol about one-half as rapidly as do slices from fed rats. This inhibition has been shown not to be due to a deficiency in alcohol dehydrogenase, nor to the DPN level, nor to a metabolic block in the oxidation of the intermediates acetaldehyde or acetate. Agents which reoxidize DPNH, such as methylene blue and ferricyanide, have been shown to increase the rate of alcohol metabolism by liver slices from fasting but not from fed rats; pyruvate and alanine raise the rate in both. DPN/DPNH ratios were found to be lowered during active alcohol metabolism in the intact animal, and this lowering was especially marked in the fasting state. Both alanine and pyruvate raised the DPN/DPNH ratio and coincidentally the rate of alcohol metabolism, with the greatest effect in the fasting rat. These results suggest that the DPN/DPNH ratio may be the limiting factor in the rate of alcohol metabolism in the fasting animal, and to some degree in fed animals. Pyruvate in the dosage employed was toxic to dogs, and produced no increase in the rate of alcohol metabolism in this species, while alanine proved quite effective, producing increases in rate of almost one-half. It is a seeming paradox that alanine, the precursor of pyruvate, is so effective in this respect, while pyruvate itself is not. The present findings do not suffice to resolve this paradox.

Vitamin K.

R. L. LESTER AND F. L. CRANE (*J. biol. Chem.*, August, 1959) have made a study of the natural occurrence of coenzyme Q and related compounds of the benzoquinone type carrying a poly-iso-prenoid side chain at position 6. It was found that many species of animals, plants and microorganisms contain this type of compound. Thus coenzyme Q is widespread in nature but not ubiquitous. As a rule, in highly aerobic tissues there occur large amounts of coenzyme Q. It is suggested that in those tissues which are apparent exceptions to this rule a different electron transport mechanism, possibly using other quinones such as vitamin K, is operative.

Thyroxine.

A. LEHNINGER (*J. biol. Chem.*, August, 1959) has studied the rapid swelling of liver and kidney mitochondria caused by L-thyroxine and related thyroactive compounds. He has shown that water can be extruded from such swollen mitochondria by treatment with ATP. The contractile mechanism is relatively stable, since addition of ATP will rapidly contract mitochondria kept as long as three hours at 20° C. following induction of swelling by addition of thyroxine. ATP is specific among all substances tested. The contraction is independent of respiration, but is inhibited by certain compounds known to inhibit phosphorylation. ATP undergoes hydrolysis during the contraction. Over 400 moles of water may be extruded per mole of inorganic phosphate formed from ATP, a finding which excludes a simple mole-for-mole mechanism for transport of water and which suggests participation of a contractile process similar to the actomyosin system.

Porphyria.

T. C. CHUE AND E. J. CHU (*J. biol. Chem.*, October, 1959) have studied in detail the urinary porphyrins of a patient with cutanea tarda. Porphyrins with five, six and seven carboxyl groups have been found, and their structures are discussed. The possibility of the existence of several isomers of the hexacarboxylic porphyrin is indicated. It is suggested that these porphyrins may be present as contaminants in copro- or uso-porphyrins prepared from biological materials by conventional methods involving extraction with ether or ethyl acetate.

Growth Factors.

I. LIEBERMAN AND P. OVE (*J. biol. Chem.*, October, 1959) report a study of the growth factors for mammalian cells in culture. A serumless medium has been prepared which supports the growth of two cultured cells of human origin, Appendix and HeLa, at rates approaching those obtained with serum. The components of the growth mixture include a partially purified serum protein (protein or flattening factor) catalase, insulin and versene (ethylenediamine - tetraacetate). With regard to insulin, the evidence is consistent with the identity of the hormone and the growth-enhancing activity. The cultured cells studied have still unknown growth requirements; indefinite multiplication does not occur in the serum-free medium, nor can colonies be formed from single cells.

Thyroid.

Y.-P. LEE *et alii* (*J. biol. Chem.*, November, 1959) have shown that the liver mitochondria of rats which are fed with desiccated thyroid oxidize L-α-glycerophosphate five times more rapidly than do normal mitochondria. This difference is not diminished by sonic disruption of the mitochondria and is still apparent in assays for the mitochondrial α-glycerophosphate dehydrogenase with phenazine methosulphate as electron acceptor. In contrast, the activity of the DPN-linked α-glycerophosphate dehydrogenase of the soluble fraction is not altered by thyroid feeding. The enhanced α-glycerophosphate dehydrogenase activity appears to involve the synthesis of new enzyme protein, for it is strongly inhibited by feeding ethionine with the thyroid-containing ration.

Histamine.

D. D. BROWN *et alii* (*J. biol. Chem.*, November, 1959) have studied the distribution of a histamine-methylating enzyme in tissues. Ring N-methylation is the principal pathway of histamine metabolism in man. Tissues on which histamine exerts its effects, i.e., skin, lung, ileum and stomach, are relatively high in N-methyl transferase activity, indicating that this enzyme can inactivate the liberated amine locally. There appears to be no obvious association between histamine levels in tissues and enzyme activity. Particularly noteworthy is the high N-methyl transferase activity in brain. Although histamine has been reported to be present in brain, no action on the central nervous system has been found to date. Histamine is the only substrate for the enzyme.

Intervertebral Discs.

E. A. DAVIDSON AND B. WOODHALL (*J. biol. Chem.*, November, 1959) have carried out analyses on herniated human intervertebral discs demonstrating changes in both the polysaccharide and protein components. The total polysaccharide content is reduced from 30% of the dry weight in normal samples to 5% of the dry weight in herniated samples. The chondroitin sulphate moiety of the polysaccharide fraction is reduced to a larger extent than is the kerato-sulphate. Ultracentrifuge patterns of herniated chondroitin sulphate indicate increased polydispersity or other degradative changes; the keratosulphate fraction appears similar to the normal. The dry weight of the herniated samples is significantly increased. A small, but reproducibly detectable amount of chondroitin sulphate B could be isolated from herniated discs. The collagen content of the herniated samples rises to 60% of the dry weight from a normal value of 30%. These changes are qualitatively similar to those seen as a result of aging.

On the Periphery.

"THE CHARACTERS OF THOSE TWO EXCELLENT PHYSITIANS, HIPPOCRATES, AND GALENUS": AN ACCOUNT OF 1659.

ON the tercentenary of a small work entitled *The True effigies, or Portraicture of the chief Philosophers, Historians, Poets, Grammarians, and Orators, or, A Compendious View of each, both dignified with, and distinguished by, their peculiar Characters,* it is of interest to draw attention to the lives of two physicians, Hippocrates and Galen, which are added, as a sort of appendix, to the philosophers. The small octavo volume was written by Ed. Larkin, A.M., "late Fellow of Kings Colledge in Cambridge, and now of Limesfield in Surrey Minister", and printed in several different sections by E. Cotes for Henry Eversden, "to be sold at his shop, at the *Greyhound* in *St. Paul's-Churchyard*". The section on the philosophers is dedicated to the Right Worshipful Sir William Haward of Tandridge, Knight, "A great Honourer of Learning, and Learned Men", to whom there is the usual fulsome dedicatory letter common in those days of literary patronage. The list of the philosophers to be discussed is given, and is followed by the statement: "Whereunto are added the Characters of those two most Eminent Physitians, Hippocrates, Galenus." The length and pattern of all the biographies are very similar, and the entries for Hippocrates and Galen, quoted below in full, are typical.

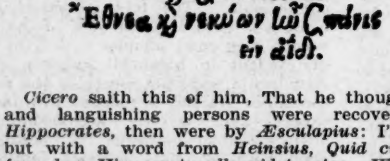
Hippocrates.

Hippocrates Cous the Son of *Heraclidas*, and the Auditor of *Democrates*, was for his knowledge in Physick far prefer'd before his own Grandfather an eminent Doctor, and of his own name; for *Suidas* gives him this honourable Encomium . . . . *The Star and Light of Physick, which is most profitable for life.* He was of great Repute with all that had ever heard of him.

Artaxerxes (as *Suidas* relates it) that mighty Emperour of the *Persians* did sollicite for, and woo his company by magnificent Largesses, and most high Advancements, as esteeming his presence equall almost to the Wealth of his whole Empire.

Cornelius Celsus calls him *Medicinae parentem virumque arte & Facundia insignem*, The Parent of Medicine, and a man famous for his Art and Eloquence, he is stiled by *Agellius, Divinia vir scientia*, A man of Divine Knowledge: By *Seneca, Maximus medicorum & hujus scientiae conditor*, The greatest of Physitians, and the Creator as it were of this Science.

Macrobius speaking also of him, hath this very passage, *Hippocratis proprium, ne fallere, aut fallisciat*, 'Tis proper to *Hippocrates*, not to know, either to deceive, or to be deceived. There is a handsome Epigram in the Greek Anthology, and it runneth thus:



Cicero saith this of him, That he thought more sick and languishing persons were recovered by this *Hippocrates*, then were by *Æsculapius*: I'le let him go, but with a word from *Heinsius, Quid cum solo conferendum* Hippocrate *ulla videt aetas.* What hath any Age seen worthy to be compared with *Hippocrates.* There were more of the Name, one a Soldier mentioned by *Thucidides.* Another a Mathematician, spoken of by learned *Plutarch.*

Galenus.

Galenus was of *Pergamus*, the Son of *Nicon*, that famous Geometrician and Architect. He is called by *Caelius Rhodiginus*, a Philosopher as well as a Physitian; who further saith of him, That in the common rumour and Fame of the World, he through his wonderfull Temperance, and Sobriety, prolonged his life to an hundred and forty years, and that he breathed alwaies Odoriferously. Whence, saith *Rhodiginus*, it became a Proverb, *Vt Galeni valetudinem dicamus pro ea, quae ultra humanum captum nimis sit prospera, nimisque inoffensa.* That we say, *Galens* Healthiness, for that, which is too prosperous, and too inoffensive beyond the naturall Capacity of men.

He flourished in the Reigns of *Marcus*, and *Commodus*; as he compiled much in Physick, so he writ also much in Philosophy, Rhetorick, and Grammar. The Learned have afforded him many eminent Eulogiums. By *Diaconus*, he is stiled *Medicus optimus.* The best Physitian, by *Casaubon, Criticorum non minus, quam medicorum princeps*, No less the Chief of Criticks, then of Physitians: By *Dempster, Medicorum, ex primo primus.*

But of all others, *Heinsius* is most high upon his Merit, who calls him, and that without flattery, *Mare eridutionis, Oceanum diciplinarum, Omnium Ingenuarum artium promum, condum, & quasi quandam Bibliothecam*; A Sea of Learning, an Ocean of Disciplines, a Butler or Drawer out of all ingenuous Arts, and as it were a certaine Library; He Composed a *Dictionary* in an Alphabetick way, upon the Learned Works of *Hippocrates*; How, and where he died, I certainly read not.

In the other biographies there are often little gems of medical information, usually in relation to the subject's death. Of Heraclitus we read that "Declining in years, he fell into a Dropsie, but would not use the help of Physitians for the curing of him. At last, tumbling himself all over Head and Ears in Dung, he was torn in pieces by greedy Dogs, or as some others say, overwhelmed, and smothered in the Dirt". Plato "dyed *Morbo pediculari*, Of the lousie Disease". Aeschylus of Athens, poet and tragedian, suffered a death both "suddain, and strange, for sitting in an open place, on purpose to decline that stroak, which by unhappy prognostication threatned [*sic*] his ruine. It fell out that, being bare-headed, and bald, an Eagle flying over him with a shell-fish inclosed in his Talons, and taking his Pate to be some stone, whereon it might crack the shell, . . . let it fall on the suddain and brained him". Other modes of death recorded are poison, broken heart, immoderate drinking and being torn apart by the barbarous and bloody women. Titus Lucretius Charus was given a "Philter" by his wife which "cast him into a Phrensie, whereof he forthwith died: whereas her only intent and design was to make him love her the better".

Bound with these works, which are continuous in pagination, is a further series of biographies, *Speculum Patrum: A Looking-Glasse of the Fathers . . .* ". The medical interest of this section is limited to several references to divine influence on the course of disease. Perhaps with truth Fulgentius observed scornfully to his physicians "Do you think that baths will hinder the death of a man by nature mortal?"

BRYAN GANDEVIA.

Public Health.

NATIONAL HEALTH AND MEDICAL RESEARCH COUNCIL.

THE following are the resolutions adopted at the forty-eighth session of the National Health and Medical Research Council held on November 12, 1959.

Resolution 1. That in relation to the uncontrolled use of synthetic dye stuffs in cosmetics the terms of reference for the Food Additives Committee should be widened to include the study of dye stuffs used in cosmetics and toilet preparations.

Resolution 2. That States should amend their legislation to make the words "HOLD UNDER REFRIGERATION" more conspicuous on the labels of canned semi-conserve hams.

Resolution 3. That in the interests of surveillance and evaluation the present requirements for the recording of poliomyelitis immunization of the individual must be continued and that the method of distribution of the vaccine must be such that it will always ensure that conditions of handling and storage will be consistent with conservation of its potency.

Resolution 4. That in view of the requirements set out for the handling, storage and recording of poliomyelitis vaccine, it cannot recommend any variation in the present

terms of issue of the vaccine to the States, until an alternative system of issue which satisfactorily meets the requirements has been devised.

Resolution 5. Where there is an outbreak of poliomyelitis which may be described as severe or explosive, the immediate commencement or the completion of poliomyelitis vaccination of all persons in the area should be undertaken.

Resolution 6. The Council recommends the use of a quadruple antigen in children under two years and, for older age groups, other appropriate antigens combined with poliomyelitis vaccine.

Resolution 7. That the Australian Atomic Energy Commission be invited to submit to the Chairman of the Council, for reference to the Radiotherapy Advisory Committee for its evaluation, proposals it has, from time to time, for the production of radio-isotopes in the form of grains, wires, rods, discs, regular and irregular shapes for radio-therapeutic purposes within Australia.

Resolution 8. That, with the reactor of the Australian Atomic Energy Commission shortly operating as a potential source of supply of radio-isotopes, the procedure for the authorization, procurement and distribution of all radio-isotopes to be used on humans in Australia should be clearly defined. It believes that authorization, procurement and arrangements for distribution should be effected through a single departmental organization.

Resolution 9. That, when an Australian source of radio-isotopes comes into operation, the well-established policies, practices and functions for authorization, procurement and arrangement for distribution continue to be, as at present, the responsibility of the Committee on Radio-isotopes of the National Health and Medical Research Council and of the Commonwealth Department of Health through its X-ray and Radium Laboratory.

Resolution 10. The Council reaffirms its policy that the use of radio-isotopes in medicine—(*a*) should be determined by clinical necessity after proper evaluation has been made of all possible methods including those not involving the use of radio-isotopes; (*b*) should be carried out only where proper facilities are available for safe handling and administration of the radio-isotopes and for evaluation of clinical and physical material; (*c*) should be carried out only where clinicians experienced with their use are available.

Resolution 11. The Council emphasizes the desirability of implementing the fluoridation of reticulated water supplies in the light of the accumulated evidence since the Council's resolutions of 1953.

Resolution 12. The Council resolved the following requirements concerning dietary supplements of fluoride: (i) Fluoride preparations should be dispensed only on prescription in order that adequate supervision may be provided for their safe and effective use. (ii) Supplementary fluorides should be prescribed only when the concentration of fluoride ion in the drinking water is known and is less than 0·7 parts per million (ppm). (iii) No more than approximately 250 mg. of sodium fluoride should be dispensed at one time. (iv) Prescriptions should be limited to those instances where the parent, or those acting *in loco parentis*, may be expected to follow directions carefully, because dietary fluoride should be continuously available throughout the period of tooth formation (at least 0-12 years of age). (v) For the guidance of medical and dental practitioners State Health Departments should be requested to publish figures where available of the fluoride content of municipal water supplies and to make these available to such practitioners.

Resolution 13. In order to prevent excessive mottling of the teeth of children and possible effects on the general health of adults, that naturally occurring potable waters containing more than 1·5 parts per million of fluoride should not be consumed habitually by children under 10 years of age and that those waters containing more than 3·0 parts per million should not be consumed habitually by adults. Where these limits are exceeded the waters should be treated to remove the excess fluoride or alternative supplies provided. In suggesting these limits the Council realizes that there are climatic and other variations determining the threshold values, but the values given have been selected as safe within these variations.

Resolution 14. Terms of Reference. The [Medical Statistics] Committee submitted for the approval of the National Health and Medical Research Council a recommendation that in addition to reporting upon matters referred to it by the National Health and Medical Research Council, the Commonwealth Department of Health and/or the Commonwealth Statistician its terms of reference shall include those specified for National Committees on Vital and Health Statistics by the World Health Organization in its Technical Report Series No. 133, page 7, namely—(*a*) To help in assessing the needs for vital and health statistics, in evaluating the degrees to which the needs are met, and in providing vital and health records and statistics satisfactory and useful to the individuals and groups who use such records and statistics. (*b*) To help to achieve essential uniformity in records, methods, and tabulations for the production of the minimum core of comparable vital and health statistics needed for national or international purposes. (*c*) To assure a free flow of information and exchange of views, so that the needs and references of producers and users of vital health records and statistics, at all levels, are given full expression and receive due consideration. (*d*) To relate the activities and functions of diverse agencies or organizations that produce statistics, so that they work as a coordinated whole, avoiding both wasteful overlapping of effort and important gaps in essential aspects of statistical data. (*e*) To make vital and health statistics of greater practical use and appeal. (*f*) To stimulate needed statistical studies by those persons or groups best able to undertake them. (*g*) To stimulate the training and supply of an adequate number of skilled workers in the field of vital and health statistics, and to encourage the interest of the medical profession in the value of the statistical approach to the problems. (*h*) To assist when desirable or necessary in the implementation of international recommendations in this field.

Resolution 15. Cancer of the Lung. (*a*) That Rubric 163 of the 7th Revision of the International Classification of the Cause of Death should only be used for the listing of those cases in which it is not possible to ascertain from the certifier whether the lung was the primary site. (*b*) The 4th Digits to Rubric 162 of the International Classification should be adopted and it is not necessary to attempt the separation of cancer of the bronchus from cancer of the lung.

Resolution 16. Classification of Type of Malignant Neoplasm. That the statistical table purporting to show the type of malignant neoplasm causing death should be allowed to lapse from the annual issue of the *Demography Bulletin.*

Resolution 17. Births. That the information recommended by Council to be collected in respect of births and fœtal deaths should be obtained from all women delivered after completion of the twentieth week of gestation or giving birth to a fœtus of 250 grammes weight.

Resolution 18. Terms of Reference. That the Council may refer to the [Veterinary Public Health] Committee any matter it considers appropriate. The terms of reference of the Committee in Veterinary Public Health may be understood to include any public health problem having a veterinary component or implication and in particular: (*a*) Any matter relating to the incidence, epidemiology, diagnosis, treatment or control of any zoonosis. (*b*) The use of animals or the products of animals as a source of food for man; the standard of purity and quality of any such food; the precaution to be taken in its production and distribution; the identification and correction of factors likely to cause such food to produce disease in man or animal whether by infection, deficiency or toxicity. (*c*) The use of animals in the preparation of biological products for use in medical or veterinary practice. (*d*) The use of therapeutic substances, antibiotics, hormones, pesticides or other substances in animal husbandry, under conditions likely, indirectly or directly, to affect adversely the health of man. (*e*) Recommendations for research projects in any one or more of these.

Resolution 19. Leptospirosis. That those States which have commenced investigations into leptospirosis on persons employed at abattoirs and other workers at special risk be asked to continue the work, and that those States which have not yet commenced investigations be asked to do so.

Resolution 20. Melioidosis. That the Queensland Institute of Medical Research be asked whether it would be prepared to undertake investigations into the ætiology and incidence of this disease for the Council.

Resolution 21. Q Fever. That States initiate or continue surveys in Q fever, and that the information obtained should be forwarded through the Commonwealth for distribution to other States.

Resolution 22. Committee Membership. That subject to the approval of the Chairman of the Council, the Veterinary Public Health Committee coopt a medical man to give advice when required on certain zoonoses.

Resolution 23. Finance. (1) The immediate provision of a supplementary grant of £21,000 to cover essential needs for 1960, thereby permitting approved projects and personnel to extend their activities through 1960. (2) That for the year 1960-61 the appropriation to the National Health and Medical Research Council should be £250,000, in order to permit the Medical Research Advisory Committee to do justice to the research needs of this country.

Clinico-Pathological Conferences.

A CONFERENCE AT SYDNEY HOSPITAL.

A CLINICO-PATHOLOGICAL CONFERENCE was held at Sydney Hospital on September 16, 1958. DR. W. L. CALOV, Honorary Consulting Physician, was in the chair. The principal speaker was MR. D. G. FAILES, Honorary Assistant Surgeon.

Clinical History.

The records of Sydney Hospital of more than 10 years ago contain the history of a young woman who had an elective appendicectomy. Eleven days after operation she had colicky abdominal pain and vomiting. The question of further operation arose.

The patient was a woman, aged 28 years who had an attack of abdominal pain three months before her admission to hospital. At first centred around the umbilicus, the pain shifted to the right iliac fossa, where it became constant and nagging. At the time she had anorexia and nausea, but no vomiting. Three weeks before her admission to hospital the pain had recurred, and there had been two similar attacks since then. Menstruation was regular and of short duration. She had frequency of micturition (passing urine seven times daily including once nocturnally), but there was no dysuria. She was in the habit of taking mild aperients, and had had pneumonia and whooping-cough and undergone a tonsillectomy as a child. Details of her reproductive life were not given.

Examination of the patient showed that she had a pale skin and conjunctivæ. Her tongue was slightly coated, her teeth were healthy and her fauces were normal. There was slight generalized abdominal tenderness not specially marked in the right iliac fossa. There was no guarding or rigidity. Rectal examination showed slight tenderness. The cardio-vascular system was normal. The blood pressure was 110/70 mm. of mercury. Vaginal examination showed a healed lacerated cervix. The specific gravity of the urine was 1·020, and it contained no sugar or albumin. At appendicectomy it was noted that the appendix was long, œdematous and inflamed, and that it was situated in a paracolic pocket.

On the day after operation the patient complained of abdominal pain, and on the second day there was an elevation of temperature to 100° F., and of her pulse rate to 110 per minute. Right-sided renal tenderness was elicited, and oral sulphanilamide therapy was commenced and continued for five days. The fever subsided next day. The bowels were open on the second, the fourth and the sixth post-operative days, and daily thereafter until the eleventh day, when the motion was liquid. On the eleventh day after operation the patient complained of colicky abdominal pain and nausea and vomited. Her abdomen was distended and tympanitic, and there was hæmatoma in and around the wound, which was discharging blood. The bowel sounds were inaudible. Gastric suction and blood transfusion were commenced. It was now thought that intestinal obstruction was possibly present, and the honorary surgeon ordered a soap and water enema, most of which was retained. His opinion then was that operation was not indicated, and that gastric suction and intravenous infusions should be continued. The temperature then rose to 102° F. and the pulse rate to 120 per minute. The chest was clear, and penicillin therapy was commenced.

At the end of the 36-hour period since the commencement of the vomiting the patient had a total fluid intake of 6800 ml. (2360 ml. intravenously), and the loss included vomitus and gastric aspirate (7200 ml.) and urine (270 ml.). Intravenous infusions of 5% glucose in normal saline solution and a 10% solution of glucose in water were given alternatively in one-litre amounts. The patient was dehydrated; the blood urea nitrogen content was 75 mg. per 100 ml. and the serum creatinine content was 4·0 mg. per 100 ml. There was no leucocytosis.

Next day the abdomen was still distended and silent. The urine was dark and highly concentrated, and contained albumin and ketones. On microscopic examination there were 10 to 15 pus cells per high-power field, and attempted culture yielded no growth. Gastric suction was continued intermittently, and the patient complained of thirst and drank frequently. During the 24 hours of the thirteenth day her fluid intake had been 6040 ml. (2500 ml intravenously), and the loss included vomitus and aspirate (5580 ml.) and urine (180 ml.). The patient would take fluid by mouth and appear to have stopped vomiting, but vomiting would recur and gastric suction would be recommenced. Firm fæces were felt in the rectum, and she passed fluid including flatus *per rectum*.

In the next 24 hours the fluid intake consisted alternately of glucose (5%) in normal saline and glucose (10%) in water by intravenous administration, and lemon drinks taken orally, the total being 6030 ml. (2250 ml. intravenously). The loss from the stomach was 3480 ml. and in fæces and urine 1230 ml. At this stage the patient appeared to be much improved. There was no vomiting, and she did not appear to be dehydrated. Bowel sounds were heard. She had a swollen, painful left arm. The position next day was apparently satisfactory. She passed a fluid motion and also 720 ml. of urine, and vomited only 6 oz. The temperature was normal and the pulse rate was elevated (110 per minute). Next day, however, abdominal pain and distension and vomiting recurred, and gastric aspiration yielded fæculent fluid. Intravenous therapy was again commenced, but the patient died a few hours later.

Clinical Discussion.

MR. D. G. FAILES: Mr. Chairman, Ladies and Gentlemen: This afternoon we are going to consider the case of a 28 year old woman who had an attack of abdominal pain three months before admission. The pain was at first centred around the umbilicus, but later shifted to the right iliac fossa, where it became constant and nagging and was associated with anorexia and nausea, but no vomiting. I think we would all agree that this is a classical history of appendicitis. The pain had recurred three weeks before admission, and there had been two similar attacks since then. Menstruation was regular and of short duration. There was frequency of micturition; this might suggest renal infection as a cause of the pain, but the absence of dysuria and the type of distribution of pain make this seem unlikely. The frequency might be due to some inflammatory process adjacent to the ureter—e.g., an inflamed appendix lying in the pelvic position. The previous history is not relevant. Details of her reproductive life were not given.

Examination showed she had pale skin and conjunctivæ. This suggests she may have been anæmic, but there is no blood count given. Her tongue was slightly coated. It has been suggested by various authors that the coated tongue, for so long a traditional sign of ill-health, is of no more significance than that the patient is a heavy smoker. We are not told if this patient was a smoker; be that as it may, it is my opinion that a coated tongue and halitosis are usually present in a case of acute appendicitis. If the tongue is clean and the breath inoffensive, then one is inclined to seek for some lesion outside the alimentary canal—e.g., gynæcological lesion. There was slight generalized tenderness, not specially marked in the right iliac fossa, and there was no guarding or rigidity. Rectal examination showed slight tenderness. Vaginal examination showed a healed lacerated cervix. The specific gravity of the urine was 1·020 and it contained no sugar or albumin.

My pre-operative assessment, then, would be that this is a case of recurrent acute appendicitis occurring in an otherwise healthy woman.

Appendicectomy was performed, and the operation findings confirmed this diagnosis—the appendix was long, œdematous and inflamed and situated in a paracolic pocket. No details of the operative procedure are given; I should like to remark that this type of appendicectomy—viz., one performed for recurrent or continuing inflammation over some weeks—may be a difficult procedure. There may be a considerable degree of local peritonitis, the tissues are œdematous, and stitches and clamps often "cut through" the tissues instead of holding; hæmostasis may be difficult to obtain, and the stump of the appendix may be impossible to invaginate. I will be interested to learn later whether or not the operative procedure in this case presented any difficulties.

We come now to the post-operative phase. On the day after operation the patient complained of abdominal pain.

On the second post-operative day, there was an elevated temperature and pulse rate associated with right-sided tenderness in the renal angle. This was interpreted as being due to renal infection and was treated with oral sulphanilamide, the fever subsiding the next day. The bowels were open normally on the second, fourth and sixth days, and thereafter daily until the eleventh day, when the motion was liquid. This fluid motion raises the possibility of a pelvic abscess. Apart from this reference to tenderness in the renal angle, there is little in the protocol to indicate why the patient was still in hospital on the eleventh day. Even those of us who do not favour early ambulation do not still have their appendicectomy patients in hospital after the seventh or eighth day. There is therefore doubt in my mind that the patient was making normal progress up to this time.

On the eleventh post-operative day there was a dramatic change. The patient complained of colicky abdominal pain with nausea and vomiting. The abdomen was distended and tympanitic, and there was hæmatoma in and around the wound, which was discharging blood. Colicky abdominal pain with nausea and vomiting, distension and tympanites suggest very strongly the presence of obstruction of the intestine. Bowel sounds were not heard, but it is not stated for what period auscultation was carried out. The presence of "showers" of turbulent, tinkling bowel sounds, coinciding with attacks of colicky abdominal pain, is of such diagnostic importance that I believe that in a case of suspected obstruction, one should observe the patient and possibly auscultate over the abdomen for at least 15 minutes if necessary.

The state of the hernial orifices in cases of suspected intestinal obstruction is so important that the results of their examination should always be noted. I presume they were clear.

Some observers now thought that there was intestinal obstruction, but the enema from which it was hoped to provide further information was retained. The honorary surgeon decided against operation, and conservative treatment was followed. However, the possibility of an inflammatory complication was strong because of the fever and tachycardia that shortly followed. The fact that the chest X-ray was clear makes it unlikely that the cause of the fever lay in the lungs.

I would like now to discuss some aspects of the case in more detail. I think we would all agree that this patient was in fact suffering from intestinal obstruction. Such obstruction must be further analysed to determine the following points: Whether or not strangulation is present? Whether the obstruction is mechanical or paralytic? The level of the obstruction? What is the pathological lesion causing the obstruction?

Further, since a good deal of this protocol is taken up with a description of the management of the case, it appears relevant to discuss the management of this common problem of intestinal obstruction in the immediate post-operative period, and also to consider the management of the fluid and electrolyte therapy and of the severe oliguria which occurred between the eleventh and thirteenth days.

For the physician, the first step in management is to arrive at an accurate diagnosis; subsequent treatment is then based on this diagnosis. For the surgeon, especially in intra-abdominal lesions, the problem is somewhat different. The first step in management may be to decide the following question: Is the patient suffering from a disease, which can and should be treated surgically? Thus the practical problem is, should operation be carried out, or not? A correct decision in this regard is more important than to countenance delay which may be involved in establishing the exact pathological cause of the emergency. An exact diagnosis is always desirable, but it is sometimes not the best practical approach. Thus, if in this patient strangulation accompanied the obstruction of the bowel, it would be of the utmost importance to operate irrespective of the cause of the strangulation, and similar remarks would apply to any mechanical obstruction. For this reason I have given prominence to the consideration of these two questions.

If strangulation had been present, the pain would have been more severe and would have been accompanied by tenderness and rigidity. Since in this patient none of these features were present, I think the possibility of strangulation is unlikely. Furthermore strangulation is curiously uncommon in the early post-operative period.

The distinction between mechanical and paralytic types of obstruction may be very difficult. Differentiation depends on an analysis of the symptoms and signs, and the following points may be helpful.

1. When pain occurs after operation, close questioning is necessary to distinguish between actual colicky pain and "soreness' in the wound. Epigastric or peri-umbilical colicky pain is typical of mechanical obstruction. However, in perhaps 50% of cases of mechanical obstruction after operation, pain is absent, and occasionally colicky pain occurs in paralytic ileus.

2. Vomiting, if copious, fæculent and persistent, suggests a mechanical obstruction. The vomiting is usually less copious and less offensive in character in a paralytic ileus.

3. As regards constipation, there are usually one or more bowel actions prior to the onset of symptoms in a mechanical obstruction. In a paralytic obstruction, there has usually been no bowel action since operation.

4. "Showers" of turbulent or tinkling bowel sounds, coincident with attacks of colicky pain, are typical of mechanical obstruction. It may take great patience to elicit this sign. The presence of bowel sounds does not entirely exclude the possibility of paralytic ileus.

A plain X-ray of abdomen showing distended loops of small bowel with fluid levels in the upright film and absence of gas in the large bowel suggests mechanical obstruction. Paralytic ileus is characterized by gross distension of both small and large bowel. The interpretation of post-operative X-ray films, however, is often difficult.

In this particular patient, bowel activity had recommenced after operation. On the eleventh day, however, she developed colicky pain, vomiting and distension; such a complication developing at this time and with these symptoms strongly suggests a mechanical obstruction. One would have expected bowel sounds to be present or even increased, but we are told they were absent. I have already pointed out that bowel sounds may be difficult to elicit, and it would be of great importance to know just how much reliance we can place on this observation. If it were true, it would favour a diagnosis of paralytic ileus.

However, it is my opinion that a mechanical obstruction is the most likely diagnosis, and certainly it cannot be excluded. I would then have no alternative but to have advised that operation be carried out after a preliminary period of resuscitation.

There are, however, many surgeons who practise conservative therapy in this type of case. Such therapy may be used in two ways. On the one hand, some surgeons use it for a trial period of 12 to 25 hours. If there is not definite improvement after this time they would advise operation. On the other hand, it may be used as a definitive method of treatment entailing intestinal intubation usually with a Miller-Abbott tube. All that I can say is that I have been disappointed with what I have seen of this method, and I feel that the risk of not operating is far greater than the risk of operating, perhaps unnecessarily in a few cases. Perhaps that is why the Miller-Abbott tube has been called the "mortal-Abbott" tube. Preoccupation with passage of the tube may obscure the necessity for operation, in cases of mechanical obstruction.

If, as I have postulated, the likely lesion is a mechanical obstruction, I think it is almost certainly in the small bowel, since vomiting and distention were early findings and onset was rapid.

We come now to the pathological diagnosis. What are the most likely causes of mechanical obstruction involving the lower small bowel eleven days after appendicectomy? Most cases of post-operative obstruction are due to lesions outside the wall of the bowel, and the commonest causes are due to adhesions, or bowel may be adherent to another segment of bowel, abdominal wall or to the wall of an abscess, as I think was the case here. There are many other causes of obstruction, but I will not even mention them, because I think there is fairly good evidence that an abscess was present in some location in this patient.

One expects, even in the presence of mechanical obstruction, that early and vigorous resuscitation should produce some amelioration in the patient's condition. It did not occur in this patient to any significant degree. Furthermore, she had pyrexia and tachycardia. I do not think the absence of leucocytosis excludes the likelihood that an abscess was present. I think this abscess was situated in the peritoneal cavity, most likely in the area of the operation. However I acknowledge that there is evidence that it may have been situated elsewhere. For example, the early post-operative renal infection followed later by

severe oliguria and right-sided renal tenderness could point to the right perinephric area. The subphrenic abscess often shows no localizing signs, and the X-ray, which might have been helpful, showed no signs at the base of the right lung.

While the liquid motion suggests the possibility of a pelvic abscess, the rectal examination makes no mention of any mass, and I think this is enough to exclude it. My interpretation of the sequence of events that occurred in this patient is as follows. Early in the second post-operative week a secondary hæmorrhage occurred, giving rise to a hæmatoma at the site of operation. The hæmatoma became infected and an abscess developed. A loop of ileum now became adherent to the wall of the abscess and became obstructed. Mounting dehydration, electrolyte imbalance and systemic effects of infection resulted in the patient's death.

I should like now to discuss the fluid and electrolyte therapy used in this patient, who was treated more than 10 years ago—before the era of flame-photometry. I think that this patient would have been suffering from severe sodium depletion. If we regard the fluid lost from the gastro-intestinal tract as roughly equivalent to normal saline, then the fluid with which it was replaced contained a much lower proportion of salt. A similar loss of potassium would have occurred for the same reasons, but may have been offset by the oliguria. The exact state of potassium balance would be difficult to assess because of these two opposing factors.

Even early in the course of the complication it was noted clinically that the patient was dehydrated. Further negative fluid balance then occurred. A significant degree of dehydration must then have been present, and this would explain the low output of urine which had a high specific gravity.

The history of this patient serves to illustrate the advances which have been made both in the estimation of electrolyte imbalances and the appreciation of their clinical value.

DR. W. L. CALOV: Mr. Failes has given us a very convincing description of the course of events in this case, which is now open for discussion. Appendicectomy is an operation which has been performed by the great majority of those present here, excluding the students; in fact, I did a few myself once. Fortunately, none of them went like this. This is now open for discussion, and first of all I would like to hear from a surgeon.

MR. R. J. MALCOLM: Looking at these post-operative problems from a slightly different point of view, it is important to bear in mind that some essential diagnosis may have been overlooked; for example, was the original inflammation of the appendix perhaps due to some contiguous inflamed viscus? Similarly, it is possible that the original condition could have been a strangulation of the bowel, and that this diagnosis might have been missed at the time of operation.

I note, on reading through the history, that there is no reference to the type of incision used in this operation. I do note, however, that there was some blood clot in the wound at a later stage. Since it is uncommon for the grid-iron type of incision to break down or give serious bleeding, we have to consider that in this case a mid-line incision may have been used. There might have been a rupture of the first line of sutures, which, of course, is always the first to give way in a burst abdomen, so that there might have been a knuckle of bowel coming through the abdominal wall.

However, I think that the diagnosis in this case is one of mechanical intestinal obstruction, consequent upon an abscess or hæmatoma. In either case, I am sure that you would have no hesitation whatsoever in operating, aspirating the contents of the abscess and, if possible, "pinching off" the loop of bowel from the abscess wall. I think I can quite truthfully say that I have known at least a dozen obstructions after appendicectomy, in which there was a knuckle of bowel on the abscess wall; this was largely in the days before antibiotics, and I just relieved the obstruction, sucked out the abscess and occasionally drained it. In all cases except one the patient recovered. So the fact that there is an abscess present in intestinal obstruction does not prevent you releasing the obstruction. The one patient that died did not actually die as a result of surgical procedures; it was a little aboriginal child, and all the trouble started up an apical tuberculosis, of which she finally died. However, I think that this must be classed as a post-operative death. One other factor of interest in this case was that the abscess was not discovered on rectal examination, although it was very large, in fact occupying the whole pelvis. I would make this warning, that you can occasionally miss a large abscess on rectal examination because you are really feeling for something smaller. However, I do not think that the abscess was situated in the pelvis in this patient.

One trick that I have in intestinal obstruction is to "rock" the patient strongly whilst auscultating over the abdomen. With the patient lying in bed, I forewarn the patient, then seize him by the iliac crest and "rock" him vigorously. In that way, you can very often discover a splashing loop when there are no bowel sounds at all. These bowel sounds may have been going on vigorously for the half hour previous to your examination, but at the time of examination you may have listened for ten minutes and not heard any bowel sounds; you must not make the deduction that it is not a mechanical obstruction of the intestines because of the absence of bowel sounds.

To sum up, I would agree with Mr. Failes that this is a mechanical obstruction on an abscess, and also I agree that the earliest possible opportunity should have been taken to operate on this case.

MR. H. H. PEARSON: Mr. Chairman, I think that Mr. Failes has given a logical answer to the problem presented by this patient, and I have no doubt that he is right. However, there are features in the history and some findings which are open to another interpretation.

There was frequency of micturition before operation, fever and renal tenderness on the second post-operative day suggested a urinary tract infection and a little later there was a sterile pyuria. It is possible that the course of events may have been that the original lesion was a ureteric obstruction followed by pyonephrosis. The appendix lying near by was associated with ureteric inflammation. On the eleventh day the pyonephrosis ruptured into the peritoneal cavity causing general peritonitis and paralytic ileus.

DR. W. E. L. DAVIES: During the long hours of the night, when residents struggle with the transfusions of patients such as this, there is a temptation, albeit not a strong one, to try to correlate the patient's progress and behaviour with biochemical findings. It may be of interest to imagine what the biochemical findings would have been if such investigations had been performed on this patient.

Findings on admission indicate that the patient had normal kidneys; she was a woman of 28 years with normal blood pressure, able to concentrate her urine to specific gravity 1·020 in a random sample, and there was no proteinuria. We are not told much of what happened in the first eleven days post-operatively, but on the twelfth day, when she had been vomiting for only 36 hours, the blood urea nitrogen was estimated at 75 mg. per 100 ml. —a figure higher than would be expected for nitrogen retention caused by such a brief period of dehydration. We are led to the conclusion that she was dehydrated for the latter part of the first eleven days.

This suggestion is confirmed by the protocol, which records that on the tenth and eleventh days she received approximately 7 litres of fluid, and that she lost approximately 7 litres, after which exchange she was considered to be dehydrated. Over the next three days the same state of affairs continued; 6 litres of fluid went in and 6 litres came out. No net gain was achieved.

Considering the above facts, it seems reasonable to postulate a deficit from the time that record of her fluid balance was kept, detectable clinically and sufficient to cause prerenal uræmia—a deficit of at least 4 litres. During treatment she was given only sufficient fluid to replace her measured losses—no allowance for insensible loss. She was pyrexial, and this insensible loss would be high—at least another 3 litres. It seems obvious that throughout the latter part of her illness she was severely dehydrated, with an uncorrected fluid deficit of at least 7 litres.

Mr. Failes has shown us a reconstructed record of her fluid balance. We saw that over the five days of this record 18 litres of fluid went in and 18 litres came out. The losses were mostly due to vomiting or gastric aspirate, and this is isotonic with the blood. She had lost 18 litres of a fluid approximating in composition to normal saline, but probably containing rather more chloride than sodium ions. Only 6 litres of the fluid given in replacement was normal saline; the remainder was glucose in water. Thus, as well as having an absolute deficiency of 7 litres of fluid (both salt and water), she also had a still greater deficiency of sodium and chloride ions. Estimation of serum electrolytes towards the end of her illness

would probably have shown a serum sodium of 120 mEq./l. and serum chloride of 60 mEq./l.

Similarly, she probably had a large deficit of potassium. Fluid motions are mentioned on the sixth to eleventh days, representing perhaps a loss of 200 mEq. of potassium. Eighteen litres of gastric aspirate represent 200 to 300 mEq. of potassium. None of this loss was replaced. She was given lemon juice. There is little potassium in lemon juice (oranges are better), and nurses cannot be induced to give patients large quantities of pure fruit juices. The patient would have received only a token dose of potassium from this source. By the final day of her illness she probably had a profound potassium deficiency—500 mEq. Estimation of her serum potassium would have been expected to reveal a level approaching 2 mEq./l. per litre.

This potassium deficiency may have contributed to the fluctuations in her condition during the last day or two of her life. On the fifteenth day intravenous fluids were stopped. She started to look better. If potassium deficiency had been making her weak, cessation of the intravenous glucose drip would have allowed a rise in the serum levels, with consequent temporary improvement in her appearance. However, vomiting made the administration of more parenteral fluid imperative. An infusion of 10% glucose in water was recommenced; her serum potassium level was again depressed. She died.

May I suggest an approach to her management? On the twelfth day when complications began, she could have been rehydrated, and her deficits of sodium, chloride and potassium corrected. With the aid of serum electrolyte estimations, this could probably have been achieved within 24 hours. She would then have been fit to undergo operation, probably the best course of action, as Mr. Failes has so clearly indicated.

Finally, at autopsy, there may be some confirmation of the above hypothesis of electrolyte disturbance: the early signs of hypokalæmic nephropathy—vacuolation of cells of the renal tubules.

DR. R. J. MALCOLM: I would like to point out that considerable fluid loss may occur into a distended bowel. One may overlook this loss, and it is rarely provided for in fluid balance records. In this patient much more severe fluid and electrolytes loss may have been present than would be apparent from the estimations given.

A doctor in the audience: It appears to me that we might be working on a wrong premise here; the loss of fluid and electrolytes may not be as great as has been supposed. This woman had a tube in her stomach and was taking fluid by mouth. The fluid aspirated would be mainly composed of the fluid that she was being given by mouth, and would not necessarily be equivalent to intestinal fluid, rich in electrolytes.

DR. W. L. CALOV: Not much attention has been paid to the finding of the bleeding from the wound. This suggests that there may have been significant bleeding into the peritoneal cavity. Mr. Failes, would you care to comment any further?

MR. FAILES: The question has been raised as to whether the aspirated fluid was only the water ingested. I do not think this is likely, since it has been shown that fluid aspirated from the stomach 45 minutes after drinking water has the approximate composition of normal saline. In answering the Chairman's question, I have already indicated that I think some bleeding occurred at the operation site. I consider this hæmorrhage as a predisposing cause for infection, and abscess formation. While I do not deny the possibility of massive hæmorrhage as terminal event, I do not think it fits in with the onset and development of the main complication.

Pathological Report.

DR. A. A. PALMER: The surgeon and pathologist who examined the appendix both noted that it was markedly œdematous. Unfortunately, from the point of view of the pathologist, the surgeon had opened the appendix; I say unfortunately, because opening destroys some of the evidence, since the contents are lost, and with them some very important evidence as to whether or not the appendix is inflamed. It is possible to have an appendix which contains pus, but which does not show definite evidence of inflammation in most parts of the wall. Furthermore, when the appendix is opened in the theatre, it is uncommon to be able to get a good section. Fortunately, in this case we have a good section. There is a very thick subserous layer, and the circular and longitudinal muscle bundles are separated far more than they should be, indicating quite an unusual degree of œdema. There are quite numerous inflammatory cells in the subserous coat—plasma cells, eosinophils and lymphocytes. These are present also in the muscle coat around a vein. Of course you can get inflammatory cells in the outer layer of the appendix from disease in adjacent organs, as Mr. Malcolm and Mr. Pearson suggest, and we have no evidence of what was inside the appendix; but I think the inflammatory changes extend so far into the muscle that this is most probably subacute or recurrent appendicitis, and it has been going on for some time because of the nature of the inflammatory cells.

DR. E. HIRST: At the time there was much anxiety about, and interest in, this patient, and the resident staff were about equally divided in their opinion as to whether operation should be performed or not. When the patient died, a post-mortem examination was at first refused, but finally permission was obtained for a limited examination, which was then performed at the funeral parlor. Our findings, though not complete, were sufficient to answer most of the questions raised in discussion today.

The abdomen was distended, and there was a little blood clot in and around the wound. When the peritoneal cavity was opened, the intestine was found to be greatly dilated and filled with fluid. There was a small amount of purulent fluid in the general peritoneal cavity, and flakes of fibrino-purulent exudate on the serosal surface of the bowel including that lying in the pelvis. A few adjacent loops of bowel were lightly adherent each to the other and were easily separated. The greater omentum was wrapped around the appendical region, and when disturbed, pus poured from this. An estimated 4 oz. of thick creamy pus was present in this abscess. No evidence of leakage of fæces from the distended caecum was found. There was no evidence of mechanical obstruction from bands, adhesions or internal hernia, and no evidence of suppurative hepatitis. I regret we were unable to proceed any further, and we have no answer to the problem of renal infection as suggested by Mr. Pearson, or possible hypokalæmic nephropathy suggested by Dr. Davies.

Our diagnosis was local peritoneal abscess in the area of recent appendicectomy for subacute appendicitis, spread into the general peritoneal cavity and paralytic ileus.

Diagnosis.

Subacute appendicitis, peritoneal abscess, peritonitis and paralytic ileus.

Out of the Past.

THE NEW SOUTH WALES ARMY MEDICAL CORPS.[1]

[From the *Australasian Medical Gazette*, April 20, 1900.]

THE numerous complimentary references which have appeared in the lay press—both in cables and in correspondence—concerning the New South Wales Army Medical Corps and its Field Hospital must be very gratifying to the profession in New South Wales. In the first place we were informed that Colonel Williams had been appointed Chief Medical Officer to all the Australasian troops, which was in itself a well deserved honour, as Colonel Williams had seen service in the Soudan and the proficient State of the New South Wales Army Medical Corps was entirely due to his energetic action. The Field Hospital of the First New South Wales Contingent under Major Fiaschi has had the honour conferred on it of being attached to Lord Roberts' headquarters staff and has received very flattering notice from the Commander-in-chief. From private, as well as from public sources we have lately learnt that Major Fiaschi was the first British officer to whom Cronje's forces began to surrender, while he was examining some bodies that were lying on the open ground between the British and Boer trenches. Major Fiaschi was also the first to enter the Boer trenches after the surrender—closely followed by Lieutenant Morgan-Martin. It has also been announced by cable that Lord Roberts has put Major Fiaschi in charge of a hospital of 500 beds at Bloemfontein.

Just as we are going to press news has been received that Colonel Williams will have charge of the medical arrangements for the new brigade of Australians and Canadians which is being formed under Major-General Hutton and that the staff will be composed entirely of Canadian doctors.

[1] From the original in the Mitchell Library, Sydney.

Correspondence.

WORLD REFUGEE YEAR.

SIR: Members are no doubt aware that this is World Refugee Year, and an appeal is being made throughout Australia for support of this worthy object. The United Kingdom Committee for World Refugee Year is undertaking this work and as some of its activities will be concerned with providing certain forms of medical assistance to refugees the matter should commend itself to all members of the medical profession. The Council of the Home Association has already promised financial support and its chairman, Dr. S. Wand, has written a letter to the *British Medical Journal* (February 20, 1960) commending the appeal to doctors in Great Britain. I have much pleasure in supporting Dr. Wand and trust that financial assistance from the medical profession in Australia for this worthy cause will be forthcoming in due course.

Yours, etc.,

H. CECIL COLVILLE,
President.

Federal Council of the British Medical Association in Australia,
135 Macquarie Street,
Sydney, N.S.W.
April 9, 1960.

GENERAL PHARMACEUTICAL BENEFITS.

SIR: We wish to record our approval of, and agreement with, the sentiments expressed in the letter by Dr. Segal and Dr. Pitts in THE MEDICAL JOURNAL OF AUSTRALIA of April 16. It becomes more obvious from week to week that the time for negotiation has indeed passed, and the time for positive action is ripe. In fact, how can one negotiate when the other party refuses to negotiate? This has been the case with the Federal Ministry of Health from the moment the scheme was implemented. Since then government by regulation has run riot, and without a blow being struck in defence we now find ourselves in the position of being unpaid clerks for the Government and reduced to economic peonage, whereby sanctions may be applied to recalcitrant members at any moment, at the whim of some lay officer in the Department of Health in the Federal or State capital concerned.

Let us take action along the lines suggested by Dr. Segal and Dr. Pitts—if necessary form our own association. After all, it only needs the majority of general practitioners in Australia to say "I won't" and the scheme collapses like a house of cards. We would be indeed interested to know how many of our colleagues actually knew of the terms of the scheme before it came into effect, and of those who did know, how many were prepared to accept it. The more facts which are bared, the more it becomes apparent that our Federal Council acted on its own initiative and its own advice, studiously ignoring those still small voices which managed to make themselves heard through the welter of political hot air.

Would it be too much to ask, through the pages of our Journal, how many of our profession are prepared to call on the Federal Council to face the Government with the wishes of the mass of general practitioners throughout the Commonwealth, and not merely make known the wishes, but inform those responsible in Canberra that either the scheme must be withdrawn and the profession allowed to draw up its own scheme—one could hardly improve on the service provided by the Department of Repatriation, for example—or let us face the government with the alternative of a complete boycott of the scheme by the profession? The third alternative is the one suggested above: if the Federal Council will not do what its members instruct it to do, out it goes, and a new Council, or the council of a completely new association, carries out the instructions of the profession.

Fighting talk maybe; but can any member of any Branch suggest anything better? Many have written letters, but so far nothing has been done, nor any suggestion made of intention to do anything, and daily we are becoming further enmeshed in the toils. Action is called for, positive action which will slash the reams of red tape in which we are in danger of strangling.

We would be happy to receive correspondence from any members in any State who would consider taking action along the lines suggested above, either through the pages of your Journal or privately. We would suggest the former, as most likely to give the politicians both within and without our ranks the shock they need.

Yours, etc.,

D. N. EVERINGHAM.
C. W. HAMMOND.
J. E. REES.

The Talbot Clinic,
P.O. Box 328,
Rockhampton, Q.
April 21, 1960.

SIR: Reading the correspondence in your Journal relating to the *Pharmaceutical Benefits Act*, I have noticed, firstly, that most of the letters of protest (I should estimate about four-fifths) come from New South Wales practitioners. Does this mean that practitioners in other States accept the additional regulations governing their prescribing with equanimity, or does it mean that they are too apathetic to protest? In either case, it bodes ill for the future freedom of the profession.

The second aspect I have noticed is that your correspondents tend to concern themselves with details of the scheme—for instance, the omission from the schedule of a specific drug, or what drugs should be provided free as doctors' bag supplies.

In discussing this whole question, let us stick to the basic principle that we must retain freedom to prescribe for our patients what we think is best for them.

The medical profession should not let themselves be involved in what is after all a financial transaction between patient and pharmacist. If the Government wishes to intervene in this transaction, then we must persuade them to do so in some way which does not involve us in a web of regulations.

Yours, etc.,

D. C. HENCHMAN.

P.O. Box 71,
Kingston, N.S.W.
April 19, 1960.

SIR: We are completely in agreement with recent correspondents to your Journal, who have condemned the B.M.A. Federal Council's supine acceptance of the new Pharmaceutical Benefits Scheme, despite mounting dissatisfaction by the profession. This is a matter affecting us all very much, and in view of the B.M.A. Council's apparent failure to appreciate members' feelings on the matter, a plebiscite should be arranged in all States, not only N.S.W., so that members may demonstrate the strength of their grievances.

Having been saddled with a scheme so unsatisfactory to us, we must energetically explain and publicize the reasons for our dissatisfaction to a public who are at the moment uninformed of the Government's duplicity. It must be made clear that the medical profession is very glad to see the Government attempting to remove the financial burden of illness from the people, and that in fact much of our opposition to the scheme is because of the anomalies and omissions which place an unduly severe burden upon many of our patients.

The medical profession should not be involved in a Pharmaceutical Benefits Scheme; it should be a matter between the pharmacists and the Government. All the restrictions with which prescribing is now hedged are merely a means by which we are forced to do much of the clerical work involved in the scheme. They are not restrictions which in any way help or safeguard the patient's interest. They take up a lot of our time in order to reduce Government clerical work. Witness the following:

1. We must write all prescriptions in duplicate on a pad of specified size and shape.

2. We may not write more than two prescriptions on a page—a third must therefore entail a second writing of patient's name and address with the date and our signature.

3. We must endorse many prescriptions "for specified purpose".

4. We may not use rubber stamps to put the date on prescriptions—everything must be in our own handwriting.

5. In order to prescribe some drugs we must obtain written permission from the Department of Health.

Besides these clerical limitations, there are restrictions on the amounts of drugs to be prescribed, the form in

which it is prescribed (the liquid form of most drugs is available only to children under ten years of age, despite the fact that many older people have great difficulty swallowing tablets or capsules), and the number of repeats permissible. These limitations, be it noted, usually bear more relationship to the cost of the drug than to the usual dosage required, and involve both extra expense for the patient to obtain the repeats necessary to provide a full course of treatment, and extra clerical work for the doctor.

One of the worst features of the new scheme is the omission of many widely accepted and frequently used drugs, e.g. "Largactil", "Butazolidin" (which is available for pensioners only), reserpine, and topical antibiotics and hydrocortisone. These are only a few of many which make absolute nonsense of the Government's statement that "practically every prescription" would be covered by the scheme. They have been guilty of shabby political dishonesty in this regard, having added to the previous free list mainly drugs which were previously cheap enough to be no severe financial burden to patients, many of which may fairly be described as archaic therapeutic drivel. It should be emphasized that the B.M.A. had no say at all in the drawing up of the list of benefits; we were not consulted.

The new scheme makes token acceptance of the principle that doctors should be allowed a small stock of free drugs for emergency use, but the small list of drugs so obtainable is quite inadequate. Again there is evidence that this list has been determined more with a view to economy than to medical requirements—e.g, procaine penicillin is the cheapest penicillin available, whereas crystalline and oral penicillin would be more satisfactory emergency drugs.

Thus we are now working a scheme which was drawn up without any consultation with the medical profession, and of which we were given no details until one week before its implementation. It places intolerable restrictions on our prescribing and involves us doing much free clerical work for the Government, and yet does not provide adequate pharmaceutical benefits for the public. In view of these facts we feel that the B.M.A. should: (i) withdraw completely from the scheme on a given date, after having given wide publicity in the medical and lay Press to the above-stated reasons for our dissatisfaction; (ii) state definitely our willingness to cooperate in a satisfactory scheme of benefits, which should provide the minimum of restrictions upon prescribing, a satisfactory insurance to the public, and of which the clerical work is undertaken by the Government; (iii) emphasize that such a scheme can only be devised by consultation between the B.M.A., the Government and the pharmacists instead of the present method of bureaucratic enforcement.

Yours, etc.,

C. BRIDGES-WEBB,
D. COLLINS,
E. L. FLEMING.

Traralgon Medical Group,
20 Kay Street,
Traralgon, Victoria.
April 7, 1960.

LIFE ASSURANCE VERSUS SUPERANNUATION INVESTMENT SCHEME.

SIR: I would like to comment on Dr. J. F. Searls' letter to you dated October 21, 1959 (published December 26), which is "off target" in several respects.

Your correspondent, in using the words "small amount of interest in the way of bonuses", implies that bonuses represent the addition (and, indeed, the only addition) of interest to the premiums paid on a life assurance policy. This view mistakenly ignores the basic fact that life assurance premiums are so calculated that, after covering the cost of the risk and the payment of expenses, the life assurance company must earn interest at some minimum rate in order that the company will be able to meet its basic obligations. Bonuses are, therefore, derived from three principal sources, first, from interest earned *in excess* of the rate assumed in calculating the premiums, second, from the claims being less than was expected, and third, from the expenses being also less than were expected.

Again, Dr. Searls claims that the life company "returns the money in pounds that are worth far less than when they were paid in". It is true that in times of inflation the proceeds of maturing policies would be lower in the purchasing power of each £1 than were some of the premiums paid, but in the same way as the purchasing power of the maturity payment would be diminished by inflation so also would the purchasing power of each succeeding premium have been diminished when it fell due for payment. The statement quoted is therefore a considerable exaggeration.

Further, that statement is made by way of comparison with the result of investment in a fund such as that of the A.D.A. scheme, which is stated to build up a capital reserve "which inflates itself as the pound is deflated, and appreciates as the cost of living rises". This statement implies that the value of that fund in terms of purchasing power will remain stationary. This is most unlikely to prove true if, as may be supposed, the assets of the fund are spread over a wide range of investment, including both "equity" investments such as ordinary shares and fixed-interest securities. "Equities" are by no means a perfect hedge against inflation; in fact "Equity" prices can move in the opposite direction to living costs as was proved in 1951-52 when the Sydney Stock Exchange index of ordinary share prices showed a *fall* from over 200 to 150 while the Retail Prices index at the same time *rose* from 1833 to 2206. It was not until 1957 that shares regained their 1951 level, during which time the price index had further inflated to 2572.

There is a further misleading statement, from which it could be inferred that life assurance premiums are not eligible for taxation concessions. It should be sufficient to state that such premiums attract concessions precisely equal to those attracted by contributions made to the A.D.A. or other similar schemes.

To claim a yield of 5½% as conservative is also, we believe, wide of the mark. The first example given by Dr. Searls of the outworking of the fund supposes that this yield will be obtained, after deduction of all expenses, year after year *for 40 years*, not only on the early contributions but also on reinvested interest and all remaining contributions. This seems to me to be anything but conservative in view of the course of interest rates over any recent period of 40 years. Historically, current interest rates are very high indeed.

What, then, is the real difference between the life assurance way and the "investment" way of providing superannuation? After an investment fund has been established for some years its pattern of investment will be similar to that of a life company, but possibly subject to different emphasis on the various types of investment. This may or may not prove to be to the advantage of the "investment" fund; time alone will tell. The only real difference at the present time is the fact that the life company fund is partly taxed but the "investment" fund is not. It seems most unlikely that, now that attention has been officially drawn to it by the Life Offices' Association, this anomaly will be allowed to continue for very long. The life companies with their unrestricted opportunities of profitable investment and their sundry other sources of surplus income can, even now, offset this disadvantage to quite an extent, and, having regard to their reputation for sound management and service, we believe that when this anomaly disappears there will be no way equal to life assurance of arranging for personal superannuation. Moreover, the life companies give guarantees, possible because of their financial size and strength, that irrespective of the state of the investment market, they will meet in full the payments under their policies and the bonuses allotted to them.

Yours, etc.,

T. P. SCOTT,
Chairman.

The Life Offices' Association for Australasia,
53-57 Collins Street,
Melbourne.
February 4, 1960.

SOME ASPECTS OF EPIDEMIOLOGY AND PREVENTIVE MEDICINE ABROAD.

SIR: May I comment on a point or two in Dr. Snow's interesting report?

Once qualified, people of many tastes and inclinations can easily be absorbed into the many branches of the medical profession, at least one of which is likely to be tailored to measure for somebody. Very few students have any clear idea at the beginning of their course about their ultimate aim. A surprsingly large number, to judge by my limited experience, are undecided even at the end of

their preregistration year of hospital residence. The undesirability of "bonding" students for later service in any particular branch of the profession is obvious; many square pegs will be forced into round holes for every one which neatly fits.

There is little doubt that preventive medicine is the least well served of the specialties in medicine. Nor is there much doubt that it is in the long run, in the ideal world, the most important branch. Of course, preventive medicine is practised by clinicians, notably by obstetricians, by pædiatricians and, to a lesser extent, by general practitioners. No doubt the practice of medicine will be nearer to perfection when all the people who visit the clinicians seek advice or treatment that will maintain their health, rather than patch up disease—when clinicians become practitioners of preventive medicine. Surely the emphasis should be placed more strongly on prevention, especially by the general practitioners of the future. Perhaps this is less dramatic than tracheotomies on the kitchen table, but certainly it is no less important. Such a realignment of emphasis as this should go a long way towards solving the problem of under-supply of specialists in preventive medicine.

At present only one Australian medical school has a department of preventive medicine for undergraduate teaching, though it is to be hoped that there will soon be others. Such departments are not strictly necessary perhaps, but they probably offer the best solution to the problem of recruitment and training of specialist workers in preventive medicine; but more important than the training of specialist workers is the need for clinicians to think more often and more positively in terms of prevention. This need will be met when preventive medicine is taught not only in the lecture theatre, but also at the bedside.

Yours, etc.,

J. M. LAST.
C.o. School of Public Health and Tropical Medicine,
University of Sydney,
Sydney.
April 20, 1960.

THE NEEDLE IN POLIOMYELITIS VACCINATION.

SIR: On a recent visit to Australia I took the opportunity of having myself and my children vaccinated against poliomyelitis. As this vaccine is not available to general practitioners, we attended vaccinating sessions held by the local municipal health authority in the area of Melbourne where we were staying. I was appalled to find that the injections were all being given from one syringe and using only one needle. The only sterilization done between injections was the waving of the needle through the flame of a spirit lamp in a vague fashion. I could not help wondering how many people developed syringe hepatitis as a result of this vaccination campaign. Surely ample evidence has been accumulated of the dangers of this sort of technique? It seems to me that in Australia there is no excuse for the continuation of this dangerous procedure. I am therefore taking the liberty of writing this letter to bring the matter to the attention of your readers, in the hope that the voice of the profession in Australia may be raised against the continuation of this practice.

Yours, etc.,

S. J. BAKER, M.D. (Melbourne),
Associate Professor in Medicine.
Christian Medical College and Hospital,
Vellore,
South India.
April 12, 1960.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

COURSES IN JUNE, 1960.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that the following programme of post-graduate courses has been arranged for the month of June, 1960.

Week-End Course in Rheumatic Diseases.

A week-end course in rheumatic diseases suitable for general practitioners will be held on June 4 and 5 in the Scot Skirving Lecture Theatre, Royal Prince Alfred Hospital. The fee for attendance is three guineas.

Week-End Course in Renal Diseases.

A week-end course in renal diseases suitable for general practitioners will be held on June 18 and 19 in the Scot Skirving Lecture Theatre, Royal Prince Alfred Hospital. The fee for attendance is three guineas.

Week-End Course in Medicine.

A week-end course in medicine will be held on Saturday and Sunday, June 25 and 26, in the Nurses' Lecture Hall, Rachel Forster Hospital for Women and Children, Redfern. The fee for attendance is three guineas. The course is open to all members of the medical profession, and limited accommodation is available for women graduates for the Friday and Saturday nights at an inclusive fee of two guineas.

Programmes.

The detailed programmes of these courses will be announced at a later date.

Course for Part I D.P.M.

A course for the Part I examination of the diploma in psychological medicine of the University of Sydney will begin on June 6 and continue for a period of eight months. Lectures and demonstrations are held in the afternoons only. The fee for attendance is 50 guineas. Information concerning requirements for the diploma may be obtained from the office of the Committee.

Course in Advanced Medicine.

A course in advanced medicine suitable for candidates proceeding to the Membership examination of The Royal Australasian College of Physicians will begin in Sydney on June 6 and continue for a period of 11 weeks. Lectures, demonstrations and ward rounds are held in the afternoons only and on Saturday mornings. The fee for attendance is 30 guineas.

Method of Enrolment.

Early written application to attend the foregoing courses should be made to the Course Secretary, The Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 4497-8. Telegraphic address: "Postgrad Sydney".

WEEK-END COURSE IN RENAL DISEASES.

The Post-Graduate Committee in Medicine in the University of Sydney announces that a week-end course in renal diseases will be held in the Scot Skirving Lecture Theatre, Royal Prince Alfred Hospital, on Saturday and Sunday, June 18 and 19, 1960, under the supervision of Dr. Ralph Reader. The Course Chairman will be Dr. T. M. Greenaway, and the programme is as follows:

Saturday, June 18: 10.15 a.m., "The Role of the Kidney in Fluid and Electrolyte Disturbance", Dr. H. Malcolm Whyte; 11 a.m., "The Role of the Kidney in Acid-Base Disturbance", Dr. Ralph Reader; 11.45 a.m., "Acute Renal Failure", Dr. David Jeremy; 2 p.m., "Urinalysis and Laboratory Tests of Renal Function", Dr. W. E. L. Davies; 2.45 p.m., "Hæmaturia", Dr. H. H. Pearson; 4 p.m., "Management of Patients with Retention of Urine", Dr. H. G. Cummine.

Sunday, June 19: 9 a.m., "The Clinical Value of Renal Biopsy", Dr. J. R. Johnson; 9.45 a.m., "Glomerulonephritis", Dr. T. M. Greenaway; 11 a.m., "Nephrotic Syndrome", Dr. R. H. Vines; 11.45 a.m., "Pyelonephritis", Dr. John McDonald.

The fee for attendance is three guineas. Those wishing to attend should make early written application to the Course Secretary, The Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephone: BU 4497-8. Telegraphic address: "Postgrad Sydney".

CONFERENCE AT ALBURY.

The Post-Graduate Committee in Medicine in the University of Sydney, in conjunction with the Melbourne Medical Post-Graduate Committee and the Border Medical Association, announces that a post-graduate conference will be held at the Albury Base Hospital on Saturday and Sunday, June 18 and 19, 1960. The programme is as follows:

Saturday, June 18: 2.15 p.m., registration; 2.30 p.m., "The Limitations of Antibiotics in Surgical Practice", Professor

M. Ewing; 3.30 p.m., "Carcinoma of the Lung", Dr. T. H. Steel; 4.45 p.m., "Medico-Legal Aspects of Practice: I", Dr. John Birrell.

Sunday, June 19: 9 a.m., "Bronchitis and Asthma", Dr. T. H. Steel; 10 a.m., "Medico-Legal Aspects of Practice: II", Dr. John Birrell; 11.15 a.m., "Methods of Promoting the Early Diagnosis of Cancer", Professor M. Ewing.

The fee for attendance at the course is three guineas. Those wishing to attend are requested to notify Dr. H. N. Meers, Honorary Secretary, Border Medical Association, 1080 Waugh Road, North Albury, as soon as possible. Telephone: Albury J.56. Arrangements have been made by the Melbourne Medical Post-Graduate Committee for their annual subscribers to attend without fee.

ROYAL PRINCE ALFRED HOSPITAL: EAR, NOSE AND THROAT DEPARTMENT.

Seminar Programme, 1960.

THE staff of the ear, nose and throat department of the Royal Prince Alfred Hospital will conduct a seminar on the second Saturday of every month at 8 a.m. in the Scot Skirving Lecture Theatre. The main speaker will not exceed forty minutes, and there will be a discussion at the conclusion of his remarks. All medical practitioners and clinical students are invited to attend.

At the next seminar, to be held on May 14, 1960, Dr. B. P. Scrivener will speak on "Malignant Tumours of the Larynx: Modern Concepts of Treatment".

Australian College of General Practitioners.

CONVENTION, MELBOURNE, OCTOBER, 1960.

THE General Practitioners' Convention arranged by the Australian College of General Practitioners, which is to be held in Melbourne from October 10 to 15, 1960, will be officially opened by His Excellency Lord Dunrossil, Governor-General of Australia, at 8.15 p.m. on Monday, October 10. At the same time His Excellency will open the associated Trade Exhibition in which over forty firms will participate.

The Academic Session of the Annual Meeting of the College at the Wilson Hall, University of Melbourne, will be opened by His Excellency General Sir Dallas Brooks, Governor of Victoria, at 8.30 p.m. on Friday, October 14.

A golf competition for the General Practitioners' Convention Cup will be held at the West Course of the Royal Melbourne Golf Club on Tuesday, October 11. This will be open to all members of the Convention. The trophy, valued at £50, has been presented for this competition by the Australian Association of Ethical and Pharmaceutical Industry.

Scientific Programme.

The scientific programme is as follows:

Monday, October 10: Reception, registration, hospitality bureau etc. Scientific tours and visits will be available to the MacCallum Cancer Institute, Commonwealth Serum Laboratories, Geriatric Units, Fairfield Hospital, Group Practices etc. by arrangement through the Convention office.

Tuesday, October 11: 9.15 a.m.: Panel and symposium on "Management of Hypertensive Diseases". Convener, Dr. Kurt Aaron (Brisbane). The panel will include a professor of medicine and a specialist physician. There will be three papers each of 15 to 20 minutes on (i) "Causes and Classification", (ii) "Symptomatology", (iii) "Management". 10.15 a.m.: Panel discussion on the papers. 11.15 a.m.: Symposium on "Low Back Pain". Convener, Dr. J. S. Collings (Melbourne). 2 p.m.: Demonstration of forensic problems, in the form of a dramatized trial around a case of tetanus in which prophylactic antitetanic serum was not used and death ensued. The trial will cover the Coroner's case and be based on the Coroner's findings, the suing of a doctor for negligence, and the implications concerning liability under the *Workers' Compensation Act*. Commentary by a member of the Victorian Bar and a Judge of the New South Wales Supreme Court. 4.15 p.m.: "Should Antitetanic Serum Have Been Given?"; and the results of a Victoria Faculty survey of allergic reactions to antitetanic serum, by Dr. Andrew Fraser.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED APRIL 2, 1960.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	2	..	..	..	..	..	..	1	3
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	3	..	1	..	..	..	10	..	14
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	1	..	..	..	1	..	..	..	2
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	1(1)	..	..	..	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	2(2)	6(5)	2(1)	..	..	..	2	2	14
Diphtheria	..	..	..	..	..	..	..	..	..
Dysentery (Bacillary)	..	3(3)	1(1)	..	1(1)	..	3	..	8
Encephalitis	..	..	1	..	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	69(30)	21(4)	10(7)	19(12)	2	1(1)	2	1	125
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	..	..	1(1)	..	..	..	1
Malaria	..	..	1(1)	..	..	..	..	..	1
Meningococcal Infection	..	..	..	..	..	..	..	1	1
Ophthalmia	..	..	..	..	3	..	..	..	3
Ornithosis	..	..	..	1	..	..	..	..	1
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	..	..	1	1	..	..	..	..	2
Puerperal Fever	..	1(1)	..	..	..	..	..	..	1
Rubella	..	11(8)	..	3(1)	1(1)	..	..	..	15
Salmonella Infection	..	..	..	..	1(1)	..	..	1	2
Scarlet Fever	9(6)	17(9)	2	3(1)	1(1)	..	..	..	32
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	1(1)	..	..	..	..	..	..	1
Trachoma	..	..	..	..	3	..	3	..	6
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	15(7)	8(6)	12(10)	8(7)	4(1)	1	3	..	51
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Wednesday, October 12: 9.15 a.m.: "Home and Holiday Hazards and their Long-Term Consequences". Convener, Dr. H. Shannon (Melbourne). Chairman, Dr. K. Bowden (Victorian Government Pathologist). "Hazards of Holidays", Dr. H. Pacy (New South Wales). 9.45 a.m.: Discussion of Dr. Pacy's paper. 10 a.m.: "Home Accident Situations", Dr. H. Shannon. 10.30 a.m.: Discussion of Dr. Shannon's lecture. 11.10 a.m.: "Socio-Economic Consequences of Accidents", Mr. McNichol Smith (Melbourne). A panel of women general practitioners, who are also the mothers of families, will be present to take part in the discussion. 2 p.m.: Annual meeting of The Australian College of General Practitioners—Business Session.

Thursday, October 13: 9.15 a.m.: "Preventive Medicine in Childhood", Professor W. Macdonald (Professor of Pædiatrics in the Medical School of the University of Western Australia). 10.15 a.m.: Discussion of Professor Macdonald's lecture. 11 a.m.: "The Scope of Research in General Practice", Dr. John Radford (Sydney). 11.30 a.m.: Discussion of Dr. Radford's paper. 11.45 a.m.: "Survey of Anæmia in General Practice", New South Wales Faculty. 2 p.m.: "Emotional Problems in Family Practice"—a new practical approach. Convener, Dr. H. Shannon (Melbourne). This will demonstrate a practical method of studying these problems in general practice, by means of a "mobile workshop" and group discussion. 4 p.m.: At Alfred Hospital Lecture Theatre, Dr. K. Bowden, Victorian Government Pathologist, will stage a demonstration of modern resuscitation methods.

Friday, October 14: 9.15 a.m.: "Can the University Prepare Us for General Practice?" A panel discussion including Professor W. Macdonald (Western Australia) and Professor Maurice Ewing (Melbourne). 11.15 a.m.: "Quiz Session' on drugs and chemotherapy, with reference to BTZ, cortisone, antibiotics, etc., with a panel to be appointed of two general practitioners, two physicians and one pharmacologist. 2 p.m. (divided into half-hour sessions): Clinical discussions will take place in the convention room at round tables on a number of topics under group leaders, certain subjects to be the responsibility of the various State Faculties. The proposed list of subjects is as follows. Virus infections, coronary disease (management), headache (New South Wales); rheumatic ailments, eczema, the crying infant (Victoria); respiratory infections in children, placental insufficiency and its influence on the infant (Queensland); abuse of antibiotics, recurrent furunculosis (South Australia); varicose ulcers (Tasmania); asthma (Western Australia).

Saturday, October 15: Victoria Faculty Pfizer Day at the Royal Women's Hospital: 10.30 a.m.: Professor B. Mayes (Sydney), lecture on obstetrical subject. 12 noon: "Post-Partum Hæmorrhage", results of a Victoria Faculty investigation. Discussion, Dr. I. L. Rowe. Commentary, Professor S. L. Townsend. 2 p.m.: Professor B. Mayes, gynæcological lecture. 2.45 p.m.: "Quiz Session", Professor Mayes, Professor Townsend and Dr. I. L. Rowe. 4.30 p.m.: Dr. David Pitt (Melbourne), a report on the results of his research into the "Effect of Rubella in Pregnancy".

In addition to this programme, a second programme will be available on various subjects at the lecture theatre of the adjacent Alfred Hospital, Melbourne, and all clinics and facilities of the hospital will be available at all times to members of the Convention.

It is also proposed that continuous or repeated demonstrations will be available concurrently with the sessions of the convention, in the following subjects: (i) trade exhibits, (ii) practice records, accountancy and taxation matters, (iii) physiotherapy demonstrations, (iv) occupational therapy, (v) social service work information bureau, (vi) films by trade exhibitors etc., (vii) research project studies.

Exhibits (i), (ii), (vi) and (vii) will be at the Convention venue. (iii), (iv) and (v) will be at the Alfred Hospital.

It is also expected that demonstrations will be arranged on various minor surgical procedures.

The programme is still subject to minor changes, and the Programme Committee would be pleased to hear from any doctor who has suggestions or requests in this matter, or might wish to participate. Communications should be addressed to the Convention Office, 132 Grey Street, East Melbourne, Victoria.

Deaths.

The following deaths have been announced:

VAN LEENT.—Johan Philip Van Leent, on April 21, 1960, at Rydalmere.

KILLINGWORTH.—Marie Jean Killingworth, on April 22, 1960, at New York.

BURNE.—Alfred Rainald Keith Burne, on April 27, 1960, at Sydney.

Diary for the Month.

MAY 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
MAY 12.—New South Wales Branch, B.M.A.: Public Relations Committee.
MAY 13.—Queensland Branch, B.M.A.: Council Meeting.
MAY 13.—Tasmanian Branch, B.M.A.: Branch Council.
MAY 16.—Victorian Branch, B.M.A.: Finance Subcommittee.
MAY 17.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAY 18.—Western Australian Branch, B.M.A.: General Meeting.
MAY 18.—Victorian Branch, B.M.A.: Clinical Meeting (Queen Victoria Hospital).

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

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ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations, other than those normally used by the Journal, and not to underline either words or phrases.

Authors of papers are asked to state for inclusion in the title their principal qualifications as well as their relevant appointment and/or the unit, hospital or department from which the paper comes.

References to articles and books should be carefully checked. In a reference to an article in a journal the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of article. In a reference to a book the following information should be given: surname of author, initials of author, year of publication, full title of book, publisher, place of publication, page number (where relevant). The abbreviations used for the titles of journals are those of the list known as "World Medical Periodicals" (published by the World Medical Association). If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

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